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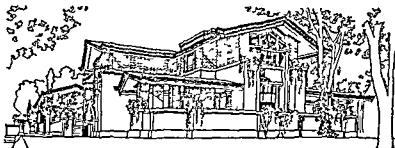
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Coronary HEART DISEASE

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CHAPTER

CORONARY HEART DISEASE

Chapter I

INTRODUCTION

CORONARY artery disease is responsible for the largest single group of organic disturbances of the heart The progressive increase in its recorded incidence during the past forty five years has been the principal factor in the rise of heart disease to the leading place as a cause of death in this country Because of its common occurrence in individuals between the ages of forty and sixty years, it affects many at the height of their careers and results in tremendous economic loss It may manifest itself in a variety of syndromes, some of which are among the most dramatic in clinical medicine The *first indication of its presence may be a suddenly fatal* attack, or it may cause various degrees of disability, up to complete invalidism for months or years before death Diagnosis may be difficult In many cases coronary disease gives rise to symptoms without causing detectable changes on physical examination or abnormalities in the electrocardio gram and in these instances recognition of the condition depends entirely upon accurate interpretation of the pa tient's complaints A considerable element of anxiety often is present and by its coloring effect on the symptoms may add to the difficulty of diagnosis Occasionally the clinical mani festations so closely resemble those of other conditions that they are erroneously attributed to a non cardiac cause The opposite type of error also may occur, and symptoms due to pathologic conditions in the pulmonary circulation aorta gastrointestinal tract, or upper spine may be wrongly charged to coronary heart disease Prognosis usually is a matter of uncertainty Although the gravity of certain signs is well established, the absence of these features does not justify

too great optimism Complications are common, and their development without warning may completely alter an outlook which until then had appeared favorable The general principles of treatment are well established, but opinions still differ as to details, and a constant search is going on for more effective drugs No measures are as yet known for preventing or retarding the pathologic changes in the coronary arteries All of these considerations combine to make this form of *cardiac disease one of the most important problems with* which the physician has to deal

The increase in the recorded frequency of coronary heart disease during the past forty five years has been attributed in part to more accurate diagnosis and in part to the greater number of people in our total population who have passed the age of forty years It has not been definitely established, however, that these factors will entirely account for the present prevalence of the condition Although coronary artery disease affects chiefly individuals beyond the age of forty years, its occurrence in younger patients seems less uncommon today than formerly The clinical manifestations in these younger people are so characteristic that there appears to be little possibility of incorrect diagnosis in similar cases in the past Other factors, in addition to improved diagnosis and changes in the population therefore, may be affecting the incidence of the disease, but there is to date no knowledge of their nature Even without them, the continued aging of our population makes it probable that this form of heart disease has not yet reached the peak of its importance

The fundamental cause of coronary heart disease is impairment of the blood supply to the heart as a result of arteriosclerosis of the coronary arteries An excellent study of this condition has been published recently by Horn and Finkelstein[1] The arteriosclerotic changes are usually distributed in an irregular manner and, in the early stages, consist of fibrotic thickening of the intima to form plaques

within which areas of lipoid deposition frequently develop The involved areas become vascularized, apparently as a compensatory reaction to maintain the nutrition of the thickened intima If new capillary channels do not develop in sufficient numbers or with sufficient speed necrotic foci form in the plaques and produce atheromatous "abscesses" Subsequently the process may go onto healing with the deposition of calcium and occasionally small areas of bone formation On the other hand hemorrhage may occur into a plaque with certain consequences which will be discussed later

The development of arteriosclerotic plaques results in areas of narrowing and at times complete obstruction in the lumen of the coronary arteries and the rate of formation of these areas is the principal factor which determines whether or not symptoms will result If the changes occur with sufficient slowness, there is opportunity for an adequate collateral circulation to develop, and the arteriosclerotic process may reach an advanced stage without clinical manifestations When the plaques evolve more rapidly, however, the collateral channels fail to keep pace, and myocardial anoxia, necrosis, and fibrosis result These changes may or may not be accompanied by distinctive symptoms Necropsy not infrequently reveals extensive coronary arteriosclerosis in persons of advanced age who had never complained of symptoms referable to the heart At times in these patients there are even one or more areas of old infarction and it may be impossible to determine whether these are the result of silent infarcts or the gradual coalescence of small areas of fibrosis [2]

Arteriosclerosis is a part of the aging process but information concerning the factors which govern the rate of its development in the coronary arteries is distinctly limited Certain diseases such as hypertension, diabetes mellitus, and conditions accompanied by a high cholesterol content in the blood serum have an important effect Approximately one

half of all patients who have coronary heart disease also have an elevated arterial blood pressure The influence of hyper tension and of diabetes mellitus is especially apparent in women below the age of fifty years, for in this group it is decidedly uncommon to encounter coronary heart disease in the absence of one or the other of these conditions

Sex is a factor of great importance in the pathogenesis of coronary heart disease Men, in the aggregate, are affected at least four times as frequently as women, and below the age of forty years the difference is many times greater than this[3] Heredity also has a fundamental bearing on the prob lem and the recent studies of Dock[4] throw light on the man ner in which this influence, as well as that of sex is exerted It has been known for some time that the portions of the coronary arteries which are affected by arteriosclerosis, namely the epicardial branches have a thicker intima than any arteries of similar caliber elsewhere in the body This is true even in the infant, and the thickness increases with age Dock believes that this disproportionate width of the intima predisposes the coronary arteries to the development of arteriosclerotic changes He observed that the coronary intima is, on the average about three times thicker in the newborn male than in the newborn female and suggested that this is the basis for the sex difference in the incidence of coronary disease The width of the intima at birth may be an hereditary characteristic, for not every newborn male infant possesses a thick intima Dock also pointed out that the presence of a thick coronary intima does not inevitably lead to the development of coronary heart disease and be lieves that arteriosclerotic changes do not occur if the arterial pressure and cholesterol metabolism remain normal

Contrary to the opinion formerly held, occupation prob ably has little if any relation to the incidence of coronary disease Levine and Hindle[5] have recently demonstrated that the condition is no more common among physicians than in

the general population Body build and temperament, on the other hand, appear to be factors of considerable importance One cannot fail to be impressed with the number of stout emotionally tense, and sensitive individuals who have this form of heart disease Whether tobacco plays a significant role in etiology has not been established, but there is a possibility that in certain persons tobacco accelerates the development of pathologic changes in the coronary arteries Much obviously remains to be learned about the factors which are responsible for the slow or fast evolution of coronary arteriosclerosis, and ultimate understanding of the problem probably will have to await the development of more advanced techniques for investigating the metabolic processes which occur in the tissues of the coronary and other arteries

The electrocardiogram frequently presents changes which are of great value in corroborating a diagnosis of coronary artery disease Alterations may be present in the P R interval, QRS complexes, RS T segments and the T waves The electrocardiogram may be normal however in patients who have advanced changes in the coronary arteries and numerous other conditions may cause electrocardiographic abnormalities similar to those found in many cases of coronary heart disease Sprague[6] recently emphasized these points and presented a long list of conditions, other than coronary atherosclerosis, which may cause abnormalities of the RS T segments and T waves The list includes the effects of several drugs myocardial and other infections pericarditis toxemic states and certain metabolic disorders changes in posture and in vagosympathetic tone axis deviation, abnormally elevated heart rates pulmonary embolism, and many other less common conditions Only myocardial anoxia and myocardial infarction produce characteristic electrocardiographic patterns which are not caused by any of these conditions These patterns will be described later No other type of abnormality in the RS T segments and T waves can be

interpreted as evidence of coronary disease unless the clinical condition of the patient is known and the various factors enumerated by Sprague have been eliminated Essentially the same restrictions apply in patients who present electrocardiographic evidence of auriculoventricular or intraventricular block

The principal clinical manifestations of coronary heart disease are angina pectoris, acute myocardial infarction, acute coronary failure, paroxysmal cardiac dyspnea (cardiac asthma), auriculoventricular and intraventricular block, other disturbances of cardiac rhythm and congestive heart failure Each of these conditions produces either a distinctive clinical picture or diagnostic changes in the electrocardiogram, and the treatment of each differs in important respects from that of the others In the pages which follow, each condition will be discussed in turn, with particular reference to diagnosis and treatment

Chapter II

ANGINA PECTORIS

ANGINA pectoris is a descriptive term applied to attacks of paroxysmal pain or discomfort in the chest which are induced by exertion or emotion and are relieved promptly by rest or termination of the emotional episode The attacks are due to relative myocardial anoxia which occurs whenever the demands upon the heart for work become temporarily greater than can be met by the available coronary blood flow [1] The anoxia probably affects one or more localized areas of the heart muscle rather than the entire myocardium, for angina pectoris is accompanied only occasionally by other evidences of impaired myocardial reserve Furthermore the development of congestive heart failure, presumably with generalized myocardial anoxia, usually results in complete disappearance of anginal attacks in patients who previously had experienced typical seizures

Pathology Angina pectoris is almost always due to coronary artery disease A few cases however are the result of other conditions such as syphilitic aortitis with narrowing of the orifices of the coronary arteries rheumatic or syphilitic disease of the aortic valve with free aortic regurgitation high grade aortic stenosis or mitral stenosis with distortion of the coronary orifices One of the most important of the recent contributions to our knowledge of coronary heart disease is that of Blumgart, Schlesinger and Davis[2] in which the clinical manifestations of the disease were correlated with the pathologic findings in the heart In their cases of angina pectoris, these authors found that, in addition to other evidence of coronary arteriosclerosis an area of old complete occlusion was always present in at least one of the major coronary arteries and usually in two of the three main vessels In all

cases, the situation was obviously ideal for the development of regional myocardial anoxia during any period of increased demand on the heart for work An old myocardial infarct was present in some of the cases, while in others no evidence of infarction was discovered In the latter patients the occlusion of the affected vessels must have occurred so gradually that there was ample time for the development of a collateral circulation sufficient to prevent the development of an infarct

Symptoms In a typical case of angina pectoris, the pain arises in the substernal region, usually in the middle or upper third, and may or may not radiate beyond this area It develops gradually, not suddenly, and becomes progressively worse until the patient is forced to stop all activity Rest gives prompt relief, the symptoms subsiding within two or three minutes in the majority of cases A desire to belch may be present, and when this is the case, the patient often is inclined to attribute his relief from pain more to getting rid of a small amount of gas than to the effect of rest An associated reflex cardiospasm or spasm of the esophagus may be present in these cases The patient frequently experiences considerable difficulty in describing the pain and often refers to it as a sensation of pressure heaviness fullness aching, tightness, burning choking, or constriction When radiation occurs, the most common distribution is to the inner aspect of the left arm down to within a short distance above the elbow Not infrequently, however, the pain extends along the ulnar aspect of the forearm to the wrist and at times to the tips of the fingers Occasionally, the upper arm is not involved and the referred pain is localized to a rather narrow zone just above the wrist In other cases there is radiation to the neck, to one or both jaws the right arm and forearm, the mid dorsal or left scapular region or to the epigastrium The pain in all of these locations is usually described as aching burning, or cramp like in character

Although the pain of angina pectoris originates in the substernal area in typical cases there are many instances in which it arises in some other region Proudfit and Ernstene[8] in a series of 500 cases found 122 (24 4 per cent) in which the pain was located in some atypical area The sites included the precordium, various parts of one or both upper extremities the epigastrium, neck throat jaws and occasionally the upper back Radiation from the point of origin to the substernal region occurred in only one third of the cases When there is no radiation of this kind, the symptoms may be attributed to some other condition unless a detailed clinical history is obtained The most important diagnostic features consist of the relation of the pain to exertion or emotion and the prompt relief afforded by rest or nitroglycerine

Other than the pain there are no constant symptoms of angina pectoris Dyspnea, palpitation, and abnormal sweating are infrequent and light headedness and syncope are very rare A sense of impending death is seldom reported or acknowledged, but many patients state that there is something about the pain in addition to its severity which forces them to cease all activity In occasional instances of far advanced coronary artery disease attacks of angina pectoris occur during sleep apparently because in these cases the *reduction in coronary blood flow during sleep is greater than* the reduction in cardiac work

Certain conditions exert an important effect upon the ease with which attacks of angina pectoris are induced by exertion or emotion Chief among these are the presence of food in the stomach exposure to cold anemia and hyperthyroidism each of which increases the amount of work the heart must do and therefore encroaches upon the coronary reserve The presence of food in the stomach and exposure to cold may also cause vasomotor changes in the coronary circulation but actual proof that such changes occur is still lacking Suggestive evidence of their occurrence is afforded by the fact that

in an occasional patient typical attacks of angina pectoris are induced by smoking cigarettes Anemia and hyperthy roidism probably cannot cause angina pectoris in patients who have normal coronary arteries The correction of these conditions, however, may so improve the coronary reserve that attacks cease to recur except with unusually strenuous exertion

There is considerable variation in the degree to which pa tients who have angina pectoris are handicapped by their symptoms Emotional and apprehensive individuals may be almost completely incapacitated by mild attacks or by the fear of inducing a seizure, while stolid patients may pay in sufficient attention to their pain and may require repeated instruction before becoming convinced of the need for keeping their activities below the point at which an attack develops

The clinical course of angina pectoris varies within wide limits in different individuals An occasional patient experi ences a few mild attacks and then remains free from symp toms for months or years Others have frequent and increas ingly severe paroxysms terminating within a short time, in sudden death due to the occurrence of ventricular fibrillation or cardiac arrest during a seizure The majority of patients suffer attacks over a period of months or years and in many the frequency of the seizures and the ease with which they are brought on fluctuates within wide limits from time to time Changes in the general health of the individual and in the outside temperature account for certain of these varia tions, while others apparently are to be explained by altera tions in the relationship between the pathologic processes in the coronary arteries and the effectiveness of the collateral circulation The development of adequate collateral channels to regions of myocardial anoxemia may afford freedom from symptoms for long periods, and an occasional patient is even permanently relieved Permanent cessation of the seizures may occur also if infarction involves an area of the myocar

dium which previously had been the site of relative anoxia In such a circumstance the region in the heart muscle which had been the source of abnormal stimuli during attacks is replaced by insensitive fibrous tissue

Signs Physical examination of patients who have angina pectoris reveals no characteristic changes either during or between attacks The heart may or may not be enlarged and its rhythm usually is regular Hypertension is present in approximately one half of the patients, and the arterial pressure often rises transiently during a seizure Pallor occasionally accompanies an attack The fixed immobility of the patient during the period of pain and the evident relief as the pain subsides may be striking features when there is an opportunity to see an actual seizure

The electrocardiogram shows changes indicative of coronary artery disease in a little less than one half of all cases of *angina pectoris* *An abnormal record in a patient who is* experiencing suggestive symptoms is helpful diagnostic evidence, but a normal tracing does not exclude the possibility of coronary artery disease The most commonly encountered change consists of inversion of the T waves in one or more of the standard limb leads or in the precordial leads, while various degrees of auriculoventricular or intraventricular block occur less frequently Records made during spontaneous seizures may show distinct abnormalities in patients who have normal tracings between attacks and in individuals who have abnormal tracings to begin with additional changes may occur during a paroxysm The most common alterations consist of depression of the RS T segments in one or more leads, diminution in amplitude or inversion of the T waves various degrees of auriculoventricular or intraventricular block, and even the characteristic pattern of anterior or posterior myocardial infarction With rest, these changes promptly disappear and the record returns to its original state

Diagnosis Since angina pectoris does not cause pathognomonic changes in physical findings or in the electrocardiogram, diagnosis of the condition depends primarily upon the proper interpretation of the patient's symptoms This calls for careful inquiry concerning all the attributes of the attacks, and there is seldom difficulty in arriving at a correct conclusion in typical cases In atypical instances and in cases where the symptoms are colored by the patient's anxiety, the chances for error are greater However, detailed analysis of the precipitating factors, location, character, radiation, and duration of the pain, and the measures which afford relief should lead to a correct diagnosis Occasionally uncertainty remains in spite of all one's efforts, and in such a situation additional help may be obtained from certain tests which have been devised during recent years These tests are based upon the known occurrence of changes in the electrocardiogram of many patients during attacks of angina pectoris The most helpful procedures are the exercise test of Riseman and Stern[9] and the anoxemia test of Levy and his associates[10] The exercise test consists of walking over a two step staircase at a standard rate of speed until pain or dyspnea develops Feil and Pritchard[11] found that the test produced positive electrocardiographic findings in 50 to 60 per cent of patients who have angina pectoris The changes which they consider significant are depression of the RS T segment of more than 1 5 mm in the limb leads and 2 mm in lead IVR, the development of the typical pattern of acute myocardial infarction or bundle branch block, or a change in the sign of the T wave except when it occurs in lead III alone The anoxemia test is performed by having a patient at rest in bed breathe a mixture containing 10 per cent oxygen and 90 per cent nitrogen until pain is experienced or for twenty minutes if pain does not occur A result is considered positive when the arithmetic sum of the RS T deviations in

failure in about one fourth After recovery from a complicating myocardial infarction, the attacks of angina pectoris usually recur as the patient resumes his former activities A certain number of individuals, however, remain free from paroxysms for a long period, and an occasional patient, as previously mentioned is permanently relieved

The development of congestive heart failure in patients who have angina pectoris is always a serious complication The attacks of pain seldom occur during the time congestive phenomena are present, probably because of the enforced rest and because the myocardial anoxia has become generalized In spite of adequate treatment including a prolonged period of rest and subsequent strict limitation of activity the patients seldom regain more than a limited myocardial reserve, and only a small number survive for two years or more

Prognosis The prognosis in the individual case of angina pectoris is always a matter of uncertainty and must always be expressed cautiously Severe and prolonged attacks which are induced by very little exertion or excitement, or occur at rest, indicate a precarious future Mild paroxysms which are experienced at infrequent intervals and are brought on only by unusual activity imply a reasonably good life expectancy but do not justify too great optimism Patients in this group usually do well if strict attention is paid to avoiding the factors which precipitate attacks but the possibility exists, nevertheless that one of the seizures will bring sudden death Not every person who has angina pectoris dies of causes related to the heart A considerable number, especially in the higher age groups, succumb to pneumonia neoplastic disease, cerebral vascular accidents, or renal failure *secondary to hypertension*

The most valuable studies on the prognosis of angina pectoris are those of White, Bland, and Miskall[14] and Parker and his associates [15] White, Bland, and Miskall reported an

analysis of 497 cases which had been followed for a minimum of twelve years Four hundred forty five patients had died at the time of the report, and the average duration of the disease in these was seven and nine tenths years The average duration of the disease in the 52 patients who were still living was eighteen and four tenths years, and by the time all of these have died the average duration of life from the onset of symptoms for the entire group will be nearly ten years These figures are distinctly more encouraging than any previously available and afford a sound basis for reasonably reassuring the patient who has angina pectoris They also indicate the measure of reward that can be anticipated from adherence to the restrictions which constitute such an essential part of the management of the condition

Parker and his associates made a follow up study of 3440 cases of angina pectoris The mortality rate for the entire group was 18 per cent during the first year after diagnosis and approximately 10 per cent annually among the survivors in each subsequent year The rate was greater for men than for women and was definitely higher for patients in the fourth decade than in those belonging to older age groups It was increased by the presence of cardiac hypertrophy, congestive heart failure hypertension, significant electrocardiographic abnormalities, and previous myocardial infarction

Treatment The most important single measure in the treatment of angina pectoris consists of having the patient do all that is possible to avoid the attacks Everything must be done slowly The habit of rushing through the morning toilet and breakfast must be corrected and there must be no hurrying to catch street cars or commutation trains Elevators should be used whenever available and stairs should be climbed deliberately Heavy lifting straining, and excitement or other emotional stimulation are to be avoided as completely as possible Usually, the patient soon learns just

what situations are liable to induce an attack, and he should be urged to regulate his life so as to eliminate these to the best of his ability An occasional individual finds the restric tions too trying for his temperament and prefers to limit his activities to a lesser degree and run the risk attendant upon a greater number of seizures Such a decision is the patient's privilege, and the physician will have fulfilled his duty if the nature of the condition has been adequately explained

Since attacks of angina pectoris are so often precipitated by less exertion soon after eating than at other times a rest period of thirty or forty five minutes after every meal is advisable Resting in a chair is sufficient and probably is to be preferred to lying down The three daily meals should be relatively small and approximately equal in size Overeating must be avoided at all times, and occasionally it is desirable to allow four or five smaller meals rather than three larger ones Overweight individuals should be placed on a reducing diet for the loss of excess weight will result in a corresponding reduction in the amount of work the heart must perform during physical activity

Because of the effect of cold on the ease with which at tacks are induced, warm clothing must be worn during the cooler months of the year, and this should include the regu lar use of gloves or mittens Freedberg Spiegl, and Riseman[16] demonstrated that the capacity of their patients to perform work without pain was greatly reduced by holding an ice cube in one hand Walking against the wind or through snow places a considerably added load on the heart and should be avoided whenever possible If the patient's economic cir cumstances are favorable, it is desirable to have him spend the winter months in the South

If anemia is present, proper treatment, including correc tion of the cause if possible should be carried out for restora tion of the blood to normal will improve the patient s exer cise tolerance and may even afford permanent freedom from

attacks The same consideration applies to the presence of thyrotoxicosis treatment by means of one of the thiourea compounds or by surgery after adequate preparation usually results in complete freedom from seizures for a considerable time or even permanently In patients who have diabetes mellitus, care must be exercised in regulating the dosage of insulin, and it often is best to be content with blood sugar levels somewhat higher than the ideal Insulin hypoglycemia is accompanied by a rise in the minute volume output of the heart, the pulse pressure, and the ventricular rate[17] These changes denote a considerable increase in the amount of cardiac work and may precipitate an attack of angina The exact role of the use of tobacco in the development and progression of coronary artery disease is not known, but there are individuals in whom paroxysms of angina pectoris can be induced by smoking a cigarette It seems advisable, therefore, to prohibit the use of tobacco in all patients who have angina except possibly those beyond the age of sixty five years In elderly individuals, the theoretical benefit to be obtained from complete abstinence seldom justifies the struggle involved in giving up a habit of many years duration Moderation should be urged, however Attacks of angina pectoris may occur while straining at stool, and because of this suitable measures should be taken to prevent constipation

Every patient who has angina pectoris should be instructed to stop all activity and stand still immediately upon the first appearance of substernal discomfort To reduce the gait without stopping is not sufficient The great majority of patients will carry out directions when specific advice is given particularly if the nature of the problem and the necessity for strict adherence to the instructions are explained One must be tactful, of course, in order not to arouse undue alarm, but it is essential that the patient understand the

significance of his symptoms Occasionally, when attacks of increasing severity are occurring at frequent intervals, rest in bed for a week or ten days is advisable

Drugs employed in the treatment of angina pectoris fall into two groups, those which are given to prevent attacks or reduce the frequency of their occurrence, and those which are administered during a seizure for relief of the pain The members of both groups act by improving coronary blood flow through a dilating effect on the coronary arteries and the newly developed collateral vessels The speed of action and effectiveness of the drugs in the first group are much less than that of members of the second but the duration of their effect is longer

Many preparations have been developed for the purpose of improving the exercise tolerance of the patient with angina pectoris and reducing the frequency of the seizures Most of these are of little actual value and none remotely approaches being an ideal drug The preparations most commonly employed are purine derivatives and include theobromine and sodium acetate, theobromine and calcium salicylate and aminophyllin It is difficult to determine the relative value of these agents but our experience has led us to give preference to theobromine and sodium acetate This is administered in enteric coated tablets in doses of 0 5 Gm ($7\frac{1}{2}$ gr) four times a day, and in many patients it seems to be distinctly helpful Since the patients daily habits have been altered at the same time and psychologic factors also are present, all of the apparent improvement in successful cases cannot be credited to the drug Nevertheless, discontinuance of the preparation for several weeks is followed not infrequently by an increase in the number of seizures and it is doubtful that this can be explained entirely on psychologic grounds Although many patients are not improved appreciably by any of the purine derivatives every individual

should be given a thorough trial on one or more of the preparations, and those who are benefited should continue to take the drug indefinitely

Alcohol in the form of an ounce or more of whiskey or brandy three or four times a day has been widely recommended as of value in the treatment of angina pectoris Many observers however, have been unable to convince themselves of its value and have discarded its use, and an explanation for this has been afforded by the recently reported studies of Stearns, Riseman, and Gray[18] These investigators found that alcohol does not shorten the duration of attacks of angina pectoris or increase the capacity of the individual for work Patients often feel better while they are taking alcohol but this is probably due to a reduction of their anxiety and apprehension, an effect which usually can be secured just as effectively from sedatives

Mild sedatives should be administered to all patients with angina pectoris who are unduly concerned about their condition or are of an excitable nature Phenobarbital in doses of 15 to 30 mg ($\frac{1}{4}$ to $\frac{1}{2}$ gr) three times a day often brings about definite improvement in the emotional state, and this may be accompanied by an appreciable reduction in the number of attacks Opiates should never be prescribed either for relief of the pain of angina pectoris or for prevention of the paroxysms The recurring nature of the seizures makes addiction inevitable if these drugs are employed

Elek and Katz[19] have reported that papaverine hydrochloride given orally in doses of 0 1 Gm ($1\frac{1}{2}$ gr) four times a day is of benefit in the treatment of angina pectoris Other investigators have not found it helpful either for shortening the attacks or for reducing their frequency, and our own experience to date has been disappointing Papaverine has been shown to produce a marked increase in coronary blood flow in the experimental animal, but the effect lasts only two or

three minutes[20] It also has a sedative action which may be a factor in its apparent usefulness in certain patients

Testosterone propionate also has been recommended as of value in improving the exercise tolerance of patients with angina pectoris The preparation usually is administered by intramuscular injection in doses of 25 mg once or twice a week for several weeks It is an effective vasodilator drug and frequently improves the patients' sense of well being Most investigators have not observed a beneficial effect on frequency or duration of attacks of angina pectoris and it is unlikely that the preparation will ever occupy an important place in the treatment of coronary artery disease

Digitalis has no place in the management of uncomplicated angina pectoris and, as a matter of fact, may make the condition worse The development of congestive heart failure or auricular fibrillation however is an indication for administration of the drug Patients in whom these complications occur should be completely digitalized, and the digitalized state should be maintained permanently by a suitable daily dose of the drug The use of digitalis also is indicated whenever a patient previously free from dyspnea reports the development of noticeable shortness of breath on less exertion than is required to induce an attack of pain The drug probably should be given cautiously as well to patients in whom the heart is enlarged even though there have been no cardiac symptoms except the paroxysms of angina If the frequency or severity of the attacks increases in these individuals the digitalis must of course be discontinued

Angina decubitus, in addition to being of ominous prognostic significance, constitutes a difficult therapeutic problem The frequency of the nocturnal attacks is seldom reduced by the oral administration of the purine derivatives Sedatives at bedtime are of help occasionally, and at times the intravenous injection of aminophyllin in doses of 0 48 Gm

(7½ gr) in the evening will enable the patient to go through the night without discomfort Dehydration of the patient by limiting the sodium chloride content of the diet and administering one of the mercurial diuretics may also be of value[11] Angina decubitus is a not infrequent precursor of acute myocardial infarction, and because of this a period of a week or ten days in bed should be advised when it first appears

The two drugs of established value for terminating attacks of angina pectoris are nitroglycerine and amyl nitrite Of the two, nitroglycerine is to be preferred Although amyl nitrite exerts its effect more rapidly than does nitroglycerine, the difference is not great enough to be of practical importance Nitroglycerine is just as effective as amyl nitrite acts for a somewhat longer time is less unpleasant, more convenient to use, and less expensive Only readily crushable tablets should be employed, and these should be broken in the mouth and held there for absorption The dose generally prescribed is larger than is necessary Tablets containing 0 3 mg (1/200 gr) usually are just as effective as larger amounts and are much less liable to cause undesirable side effects such as headache or throbbing in the head, dizziness, flushing of the face, and heart consciousness Every individual who has angina pectoris should be supplied with a number of the tablets with instructions to use one whenever an attack is not relieved by rest within one minute A few patients hesitate to employ the drug either because it causes unpleasant symptoms or because of a mistaken idea that harmful effects may result from its use The first of these objections usually can be overcome by prescribing tablets of smaller size, and the second by reassurance The repeated use of nitroglycerine is not detrimental, and because it is important that every attack of angina pectoris be brought to an end as quickly as possible all patients should be urged to employ it without hesitation The fact that the drug is available and is not

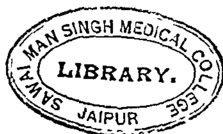
injurious does not excuse a person from exercising every possible precaution to avoid attacks

Nitroglycerine is used not only for the relief of anginal pain *but also as a means of preventing the development of* seizures Many patients are forced by the nature of their occupation to perform tasks which regularly precipitate a paroxysm of pain, and in these persons the attacks often can be prevented by taking nitroglycerine shortly before beginning the unavoidable exertion In unusual circumstances from 12 to 20 tablets may be used in this manner each day When seizures are liable to follow a meal, the drug may be given for its prophylactic effect either before or immediately after eating The frequent use of nitroglycerine in this manner does no harm and often enables the patient to get along comfortably and remain self supporting for a considerable length of time It does not relieve him, however, of the responsibility for avoiding unnecessary activities that may bring on an attack

Although nitroglycerine affords prompt relief from paroxysms of angina pectoris which are induced by exertion or emotion, *it often has little or no effect on attacks of angina* decubitus Patients who are suffering from this form of angina *frequently find that in order to obtain relief they must sit* up or stand The pain may subside promptly after the change in position

In patients who are unable to perform activities of any kind without inducing an attack of angina or who experience seizures while at rest or asleep, some form of surgical treatment may become advisable The most commonly employed procedures are paravertebral injections of alcohol to destroy the upper four or five thoracic sympathetic ganglia, surgical removal of the upper four or five thoracic sympathetic ganglia, and laminectomy with section of the upper four or five thoracic posterior roots [21] Alcohol injection is the simplest

of the three measures but is more liable to give an unsatisfactory result than the other two methods of treatment A painful intercostal neuritis results in a high percentage of cases and may last for many weeks or months There is also danger of other complications such as pleurisy, pneumothorax, and toxic myelitis Furthermore, attacks of angina may recur as the effects of the alcohol wear off Sympathetic ganglionectomy is a much more precise procedure and is the surgical treatment of choice in cases in which the pain of the attacks is unilateral in distribution When the pain is strictly substernal, however, or when it radiates both to the right and left laminectomy with bilateral rhizotomy should be recommended According to Ray,[21] this procedure involves little more risk than a sympathectomy, and it has the distinct advantage of being a bilateral operation that can be completed in one stage Surgical treatment is necessary in only a small number of patients with angina pectoris, and it should be advised only in extreme cases and only after all other measures have proved ineffective



Chapter III

ACUTE MYOCARDIAL INFARCTION

THE SYNDROME of acute myocardial infarction is one of the most dramatic, dangerous, and highly publicized of the manifestations of coronary heart disease. The first complete description of its clinical features was published by Herrick[22] in 1912, and a voluminous literature dealing with all its aspects has developed since that time. The incidence, pathology, symptoms, and signs of the condition have been investigated extensively, and the accumulated information has been widely disseminated. Diagnosis has become a matter of great accuracy, and increasing knowledge of the electrocardiographic changes to which the condition gives rise has made it possible to localize an area of infarction with remarkable precision. Recognition of the frequency of the condition has corrected the earlier impression that the initial attack usually results fatally. There is general agreement concerning the essential features of treatment, and the recent introduction of anticoagulant therapy has raised the hope of further improvement in therapeutic results. The present state of our knowledge of myocardial infarction is therefore favorable, but much still remains to be learned.

Etiology. The etiology of coronary artery disease in general has been discussed earlier, but a few additional points which apply specifically to acute myocardial infarction should be mentioned. This form of coronary heart disease affects men approximately six times as often as women and occurs predominantly in individuals more than fifty years of age. However, it appears to be increasing in frequency in patients belonging to lower age groups. Acute infarction may occur as the first manifestation of coronary artery disease, but

approximately one half of all patients have experienced earlier attacks of angina pectoris, and many others have had dyspnea or other evidence of reduced myocardial reserve Severe or unusual exertion, intense emotion, over eating, or exposure to cold may be directly responsible for the onset of the attack At times, shock, hemorrhage, paroxysmal tachycardia, or other factors which reduce coronary blood flow precipitate the symptoms In the majority of cases, however, the condition has its onset while the patient is at rest, and in an impressive number the initial symptoms waken the patient from sleep

Pathology The left coronary artery, through its anterior descending branch, supplies the anterior wall of the left ventricle, the adjacent one third of the anterior wall of the right ventricle, and the anterior two thirds of the interventricular septum [3] The other main division of the left coronary artery, the circumflex branch, supplies the lateral margin and from one third to one half of the posterior basal portion of the left ventricle The right coronary artery supplies all of the right ventricle except the medial one third of the anterior wall, and, in addition, is distributed to the posterior third of the interventricular septum and the medial one half to two thirds of the posterior, basal portion of the left ventricle Attention has been directed repeatedly to the fact that this anatomic distribution of the coronary arteries is subject to wide variation Blumgart and Schlesinger[2] divided their cases into three general groups, one with a balanced coronary circulation and the other two with right and left coronary artery preponderance respectively They found that in cases of left coronary artery preponderance, arterial occlusion caused a higher incidence of myocardial infarction than in the other two groups, and the mortality rate of the acute attack was much greater

In general, sudden occlusion of the anterior descending branch of the left coronary artery results in infarction in the

anterior apical portion of the left ventricle with or without involvement of the interventricular septum, while occlusion of the circumflex branch causes infarction in the postero lateral or posterior basal portion of the left ventricular wall Sudden obstruction of the right coronary artery is followed by infarction of the posterior basal portion of the left ventricle Infarction confined to the right ventricle alone is extremely rare It was believed formerly that occlusion of the anterior descending branch of the left coronary artery was much more common than occlusion of the circumflex branch or the right coronary artery, but within recent years several investigators have demonstrated that this is not the case Involvement of the anterior descending branch or its primary divisions is only a little more frequent than is occlusion of the right coronary artery Obstruction of the circumflex branch on the other hand, is much less common Since occlusion of the right coronary artery or the circumflex branch of the left results in posterior basal infarction, the incidence of this type of infarction is approximately equal to that of anterior apical infarction

Several observers have directed attention to the fact that when myocardial infarction is found at necropsy areas of occlusion frequently are present in two or more coronary branches Horn and Finkelstein[1] for instance, observed simultaneous occlusion of two or more arteries in 48 of 100 cases of acute infarction and Saphir and his associates[24] found complete occlusion or extreme narrowing of two or more branches supplying the infarcted area in all of 31 cases The latter investigators also pointed out that while the infarct usually is situated in the region supplied by the involved coronary arteries acute occlusion of one artery may cause infarction in a remote area supplied by a vessel that had been occluded at an earlier time In these cases the nutrition of the myocardium is maintained after the first occlusion by collateral anastomoses, and infarction does not

occur until the vessel which is the source of the collateral circulation becomes obstructed The same mechanism undoubtedly accounts for certain of the cases in which two areas of acute infarction occur simultaneously

Acute occlusion of a coronary branch may result from hemorrhage into the intima of a sclerotic artery, from the formation of a thrombus on an arteriosclerotic plaque, or from an embolus dislodged from a thrombus in the proximal portion of a coronary artery Horn and Finkelstein[1] found that the first of these mechanisms is the most common and causes coronary artery occlusion in one of three ways The intramural hemorrhage frequently induces acute degenerative and reactive changes in the overlying plaque, and these changes are followed by the local development of a thrombus Secondly, the hemorrhagic area may rupture into the lumen of the vessel and thus afford a focus for thrombus formation Finally, an intramural hematoma may, in rare cases, attain sufficient size to cause mechanical obstruction of the vessel without rupture of the intima When a thrombus forms on an arteriosclerotic plaque showing no evidence of intramural hemorrhage, degenerative or irritative changes usually are present in the subendothelial tissue and are responsible for the development of the occlusive lesion

Occlusion of a coronary artery does not always cause myocardial infarction The rate at which the obstruction develops and the presence or absence of an adequate collateral circulation are the factors which determine whether or not an area of necrosis will result Pathways for collateral circulation are present in the normal heart but under ordinary circumstances are of little or no functional significance In the presence of extensive coronary disease, however, they may become an important or even the sole source of blood supply to the myocardium Leary and Wearn[25] reported 2 cases of complete occlusion of both coronary orifices with no evidence of infarction, and in these cases an adequate blood

supply must have been maintained entirely through col lateral channels

The collateral pathways which, under conditions of neces sity, may assume an essential role in maintaining the cir culation to the myocardium consist of thebesian veins, arteriolumınal and arterio sinusoidal vessels, intercoronary anastomoses, and extracardiac communications The the besian veins are small vessels which connect the cavities of the auricles and ventricles with the coronary veins and the capillary bed of the myocardium The arterio luminal and arterio sinusoidal vessels were first demonstrated by Wearn and his associates [6] and differ from the thebesian veins in that they connect the cavities of the heart with the arterial instead of the venous side of the coronary system The arterio luminal channels are short vessels of the size of arterioles which run directly between the heart chambers and coronary arteries or arterioles lying near the endocardium The arterio sinusoidal vessels arise as branches of a coronary artery or arteriole and quickly break up into irregular channels, from 50 to 250 micra in diameter These are myocardial sinusoids and they empty into the cavities of the heart The rarity of infarction involving only the right ventricle may be due to the fact that the thebesian veins and the arterio luminal and arterio sinusoidal vessels are able to maintain an ade quate circulation to the thin wall of this ventricle when called upon to do so

The extracardiac communications consist of anastomoses *between the coronary arteries and arteries* in *the mediasti* num, lungs parietal pericardium and diaphragm [27] Collateral channels also exist between the coronary arteries and the vasa vasorum of the aorta It is doubtful that these extra cardiac anastomoses ever spontaneously assume a role of clinical significance

The most important collateral pathways in the myocar dium consist of anastomoses between branches of one coro-

nary artery and those of another The existence of these intercoronary vessels is well established but there is some disagreement as to their maximum diameter in normal individuals Blumgart and Schlesinger[2] were unable to demonstrate vessels of this type with a diameter greater than 40 micra in normal hearts but observed that arteriosclerotic narrowing or occlusion of coronary arteries regularly resulted in the development of channels measuring from 40 to 200 micra in diameter The development of these larger anastomoses was found not to be related to the age of the patient, for they were not present in the hearts of elderly individuals who had little or no coronary arteriosclerosis Prinzmetal and his associates [26] on the other hand, reported the existence of intercoronary anastomoses with diameters of 70 to 90 micra in 8 of 13 normal hearts and 160 to 180 micra in 3 of the remaining 5 In general agreement with Blumgart and Schlesinger, however, they found that the maximum size of these vessels in the presence of coronary arteriosclerosis ranged from 120 to 150 micra It is apparent that collateral channels of the latter size should be able to compensate to an important degree for marked narrowing or complete occlusion of a major coronary artery An anatomic reason therefore exists for the frequent failure of thrombotic coronary occlusion to result in myocardial infarction

Although the most common cause of myocardial infarction is acute coronary thrombosis, it must be emphasized that myocardial infarction and coronary thrombosis are not synonymous terms Not only may coronary thrombosis fail to cause myocardial infarction, but infarction not infrequently occurs in the entire absence of an occlusive arterial lesion Friedberg and Horn[29] found no evidence of recent coronary occlusion in 31 per cent of their autopsied cases of acute infarction Infarction in cases of this kind, is due to some factor other than an obstructive lesion which causes prolonged anoxia of the myocardium Frequently the cause

is a sudden increase in the demands on the heart by exertion emotion, overeating, exposure to cold, or a disturbance in cardiac rhythm accompanied by a rapid ventricular rate In other cases, an abrupt and prolonged reduction in coronary blood flow secondary to hemorrhage traumatic or surgical shock or a fall in arterial pressure due to spinal anesthesia is responsible Myocardial infarction can be diagnosed with great accuracy, but often one can only speculate in a given case as to whether the condition has resulted from acute coronary thrombosis or from prolonged myocardial anoxia without coronary occlusion

The histologic changes which occur in an area of myocardial infarction have a direct bearing on the treatment of the condition Mallory White and Salcedo Salgar[30] found that the prevailing changes during the first week are necrosis of muscle and infiltration by polymorphonuclear leukocytes, while removal of the necrotic muscle and replacement by connective tissue predominate during the next five weeks Small infarcts were observed to be almost completely healed after five weeks and large ones after two months The relation between these findings and the advisable duration of bed rest after myocardial infarction is obvious

Myocardial infarction usually results in some enlargement of the heart although in many cases this is of such limited degree that it cannot be demonstrated by clinical or roentgen ray examination Bartels and Smith[31] found gross cardiac hypertrophy in 37 of 42 autopsied cases of myocardial infarction in which no other known cause of hypertrophy was present The average increase in the weight of these hearts above the estimated normal was 132 grams (44 per cent)

Symptoms The onset of acute myocardial infarction often is preceded at an interval of several hours to a few days by certain premonitory symptoms and occasionally these antedate the acute attack by as long as three weeks Attention was first directed to their occurrence and signifi

cance by Feil[32] and Sampson and Eliaser[33] Feil estimated that they are present in approximately 50 per cent of all patients, and Master, Dack, and Jaffe[34] reported an incidence of 44 per cent in 260 cases Master and his associates attributed the symptoms to gradual occlusion of a coronary artery by progressive or recurrent intramural hemorrhage or by the development of a thrombus on an arteriosclerotic plaque Blumgart, Schlesinger, and Zoll[35] pointed out, however, that they may be due to the syndrome of coronary failure which will be discussed later Correct interpretation of the symptoms is important because the prompt enforcement of strict rest in bed may prevent the occurrence of infarction when the symptoms are the result of coronary failure When the symptoms are due to gradual coronary occlusion, rest probably will not arrest the further development of the thrombus and avoid infarction, but the early treatment of infarction in this manner should reduce the mortality rate

Premonitory symptoms are of different types Perhaps the most common consists of the sudden appearance of severe attacks of angina pectoris in a patient who previously had experienced no symptoms referable to the heart In individuals who have had earlier attacks of angina, an abrupt increase in the frequency and severity of the seizures has the same significance Other patients, who may or may not have had angina pectoris, experience a sudden attack of severe substernal pain which lasts for more than twenty minutes and is only partially relieved by nitroglycerin This is due to acute coronary failure Occasionally the pain is only mild or moderate in degree but lasts more or less continuously for as long as forty eight hours In still other instances premonitory pain is absent, and the patient complains only of increasing weakness and fatigue

The actual onset of the attack of acute myocardial infarction is characterized, in all but a few cases, by the appearance

of pain similar to that of angina pectoris but generally more severe and almost always of longer duration The pain usually is situated beneath the upper or middle third of the sternum although it arises beneath the lower third or in the epigastrium more often than does the pain of angina pectoris When it originates in the epigastrium or some other area of the upper abdomen, it almost invariably spreads promptly to the substernal region and in those cases in which it originates in the chest, radiation to one or both arms the neck or jaws is common The pain is described by different patients as a feeling of fullness, pressure gas tightness or heaviness or as a choking, burning or crushing sensation The discomfort may be intense at the very onset but in most cases it comes on gradually and increases progressively in severity Nitroglycerin is without effect and morphine gives only gradual relief The pain almost always lasts for more than one half hour and usually for one to several hours the determining factors being the size of the infarct, the promptness with which treatment is instituted, and the degree of accompanying shock Profound shock may quickly render the patient insensitive to all painful stimuli

Symptoms of shock of mild to severe degree appear soon after the onset in practically all patients Cold, clammy perspiration, weakness and faintness are common, and at times there is extreme vasomotor collapse Nausea of some degree is usually present, and in a certain number of cases there is repeated vomiting

Although pain is the predominant symptom in the great majority of cases it is entirely absent in a small group of patients In some of these its absence is due to traumatic or postoperative shock, pre-existing congestive heart failure or depression of the sensorium by drugs or toxemic states but in others no such factors are present When there is no pain the occurrence of infarction may be manifested by syncope, collapse, sudden extreme weakness, or the symptoms of acute

left ventricular failure Sudden weakness, syncope or collapse in a person more than forty years of age always should suggest the possibility of acute myocardial infarction although some other cause usually will be found to have been responsible The onset of acute infarction generally is accompanied by a certain amount of respiratory distress, and at times this is of severe degree In painless attacks, sudden intense dyspnea, with or without asthmatic breathing, may overshadow all other symptoms and may be followed promptly by acute pulmonary edema When myocardial infarction occurs during the course of congestive heart failure, the only indication of its onset may be an abrupt increase in dyspnea and in the evidence of congestion in the pulmonary or systemic circulation

Signs Physical examination shortly after the occurrence of acute myocardial infarction usually reveals an apprehensive individual in obvious distress The skin is moist, cool and clammy, and ashen pallor or definite cyanosis frequently is present The pulse is generally rapid and weak, but occasionally, because of reflex vagal inhibition of the heart or a high degree of auriculoventricular block, it is abnormally slow The arterial blood pressure may rise transiently with the onset of the pain but soon falls below its original level In many patients, no initial rise is observed and the appearance of symptoms is accompanied by an abrupt drop of considerable magnitude In still others, and especially in those who have hypertension, the reduction may not amount to an appreciable figure for three or four days, and in most cases the lowest pressure is not reached until the third week The heart tones are of poor quality, gallop rhythm is often present, and the cardiac rhythm may be irregular because of premature beats, partial heart block or auricular fibrillation A pericardial friction rub develops during the first few days in approximately 10 per cent of all cases and may last for a few days or only a few hours Signs of congestive heart failure may appear promptly after the onset or at a later date

Fever and leukocytosis usually develop within twenty four hours of the onset, and the erythrocyte sedimentation rate becomes elevated within the first three or four days These features are due to necrosis of myocardial tissue and their magnitude and duration vary according to the size of the infarcted area The average maximum temperature is about 101° F , but fever of greater degree is not uncommon The leukocyte count usually reaches a peak of 12 000 to 15,000 cells per cu mm and occasionally rises to 25 000 or more The erythrocyte sedimentation rate remains elevated much longer than the temperature or white blood cell count

The electrocardiogram shows diagnostic changes in practically every case of acute myocardial infarction if records are made every few days and if precordial leads are used in addition to the standard limb leads Significant changes may appear immediately after the onset of symptoms or may not develop for several days At times only minor abnormalities occur, but when these are compared with the findings in earlier records or when they shift from day to day they are sufficient to corroborate the diagnosis In general, the electrocardiographic findings are determined by the size and location of the infarct and by whether one or more areas of acute infarction are present The record may be greatly affected, however, by such complicating factors as left bundle branch block an old, healed infarct, or diffuse pericarditis Two principal electrocardiographic patterns have been recognized for several years [36,37,38,39] and it recently has become possible to subdivide these by the use of multiple precordial leads [40] All of the patterns go through orderly changes as healing occurs and if the infarct is not too large the record may return to normal after a period of weeks or months The two main patterns are those of anterior apical and posterior basal infarction

In acute anterior apical infarction the most important electrocardiographic changes occur in lead I The earliest of

these consist of the appearance of a Q wave, a decrease in the R wave and elevation of the RS T segment When the elevation of RS T is marked, the T wave may not be discernible In lead III a reciprocal depression of the RS T segment occurs and may obscure the T wave Prominent S waves appear in leads II and III These early changes last for only a few hours or days, after which the RS T segments return gradually or rapidly to, or almost to, the iso electric level Coincident with this, the RS T segment in lead I becomes upwardly convex and is smoothly continued into a prominent, sharply inverted T wave (Pardee's sign[41]) At the same time the Q wave in lead I increases in amplitude, and in lead III a sharply peaked positive T wave develops During the following few weeks the convexity of the RS T segments gradually disappears and the T waves become less prominent and more rounded The Q wave in lead I usually persists, although it frequently becomes smaller After weeks or months, the T wave in lead I may become upright, and Q_1 may disappear at the same time or subsequently The record may then present no changes to indicate the former occurrence of anterior apical infarction

Acute posterior basal infarction is characterized by electrocardiographic changes which are the reciprocal of those that occur in anterior apical infarction Conspicuous Q waves appear in lead III the RS T segments become elevated in lead III and depressed in lead I, R_3 decreases in amplitude and the T waves may be obscured in leads I and III Within a short time the RS T segments return to, or almost to, the iso electric level and in lead III they become upwardly convex A sharply peaked, inverted T wave develops in lead III and a correspondingly sharp but upright T appears in lead I Later, the RS T segments lose their convexity, and only a prominent Q_3 and an inverted but no longer peaked T_3 remain as evidence of the infarct Eventually even these changes may disappear Throughout the entire cycle lead II usually shows

abnormalities similar to but of lesser degree than those in lead III

If multiple precordial leads* are employed it is possible to localize the site of infarction more accurately than can be done by the use of limb leads alone Leads of the unipolar variety are to be preferred Patterns indicative of antero lateral, anteroseptal, high lateral, plain posterior postero-lateral and postero inferior infarction have been identified [40] The differentiation between these various patterns is made possible by the fact that the most marked diagnostic changes occur in the precordial leads which are made with the ex ploring electrode directly over the site of infarction As the electrode is moved away from the infarcted area, the ab normalities rapidly become less pronounced and in leads made at a distance from the involved region, only slight or no alterations are present In an individual case, therefore the number of standard precordial leads which show char acteristic changes depends on the size of the infarct and its proximity to one or more of the positions specified for the exploring electrode The abnormalities which are diagnostic of myocardial infarction consist of disappearance or great diminution in amplitude of the R waves, the appearance of prominent Q waves pronounced upward displacement of the RS T segments, and sharp inversion of the T waves The elevation of the RS T segments ordinarily lasts for not more

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* In recording multiple precordial leads records are made with the exploring electrode at each of the six points recommended by the Committee of the American Heart Association for the Standardization of Precordial Leads.[41] The first of these points is in the fourth intercostal space at the right margin of the sternum and the second is in the same interspace at the left margin of the sternum. The remaining points lie on a line drawn from the second point to the apex beat and then continued around the left side of the chest at the level of the apex. When the apex beat cannot be located the line is drawn from the left sternal border in the fourth intercostal space to the point where the left midclavicular line crosses the fifth intercostal space and is then continued around the chest at the level of this intersection. The third point is midway between the second and the left midclavicular line the fourth is in the midclavicular line or at the apex beat the fifth is in the anterior axillary line and the sixth in the midaxillary line.

than a few days but the changes in the QRS complexes and the inversion of the T waves may persist indefinitely

In anterolateral infarction, the leads from the left side of the precordium (V_4 and V_5, and at times V_6) show diagnostic changes, and these usually are accompanied by the characteristic pattern of anterior infarction in the limb leads

Anteroseptal infarction is characterized by diagnostic changes in the chest leads from the right side of the precordium, especially V_2 and V_3 The limb leads show alterations suggestive of anterior infarction but these often are not sufficiently developed to be diagnostic

In high lateral infarction the limb leads present changes characteristic of anterior infarction but the standard precordial leads show no diagnostic signs If the chest leads are moved up one or two intercostal spaces, however, typical changes will be found in the leads from the left side of the precordium

Plain posterior infarction causes the characteristic pattern of posterior infarction in the limb leads The R and T waves in the leads from the right side of the precordium may be exceptionally prominent, and in the earliest stages there is downward displacement of the RS T segments in all precordial leads The depression of RS T usually is of short duration, and the precordial leads otherwise show no diagnostic changes Esophageal leads made at the ventricular level present characteristic signs of infarction

Postero inferior infarction, like plain posterior infarction, produces changes in the limb leads diagnostic of posterior infarction Unlike plain posterior infarction, however, the standard precordial leads show no significant abnormalities, and the R and T waves in leads V_1 and V_2 are small rather than unusually tall Diagnostic changes may be present, however, in a lead from the left border of the tip of the ensiform cartilage

In posterolateral infarction characteristic signs are present

in the left lateral precordial leads (V_5 and V_6), and the R and T waves in leads V_1 and V_2 are abnormally prominent The limb leads show either changes consistent with posterior in farction or sharply inverted T waves in lead I and prominent Q waves in leads II and III Wilson and his associates[40] have pointed out that the latter pattern in the limb leads may also result from a combination of an old posterior infarct and a more recent anterior one, and at times from anteroseptal in farction

The electrocardiogram in acute myocardial infarction may present other abnormalities in addition to the changes char acteristic of the various types of infarction, and these may obscure the above diagnostic patterns Partial or complete auriculoventricular block may result from involvement of the basal portion of the interventricular septum but, of course, will have no effect on the changes in the QRS com plexes, RS T segments and T waves Intraventricular block of any type may be present Right bundle branch block does not mask the picture of infarction but in left bundle branch block the changes due to infarction are obscured The de velopment of acute pericarditis as Barnes[43] originally pointed out, alters the reciprocal relationship between the RS T segments in leads I and III Instead of an elevation of RS T in one of these leads with a corresponding depression in the other, the egments are displaced upward in all three limb leads A typical Q wave pattern may be present however and a corresponding sharp inversion of the T waves may de velop later

Differential Diagnosis Early writers[22,44] directed attention to the fact that acute myocardial infarction may closely simulate biliary colic or acute surgical conditions in the upper abdomen, and the possibility of erroneously attributing the symptoms of infarction to upper abdominal disease has since been emphasized repeatedly Within recent years the pos sibility has been pointed out that errors also may be made in

the reverse direction, and cases have been reported in which symptoms due to gall bladder disease or perforated peptic ulcer suggested myocardial infarction Pulmonary embolism and dissecting aneurysm of the aorta likewise give rise to clinical pictures which may be confused with that of acute infarction

The most helpful guides in the differentiation of biliary colic or acute surgical conditions in the upper abdomen from myocardial infarction are the presence of localized or generalized splinting of the abdominal wall, localized tenderness or a palpable mass in the abdomen, failure of the pain to spread to the substernal region, absence of abnormalities of the heart sounds, and the presence of a normal electrocardiogram Although the pain of acute myocardial infarction may originate in the upper abdomen, it almost invariably spreads from that area to the substernal region within a relatively short time Radiation of this kind is extremely unusual in painful conditions in the abdomen

Embolic occlusion of the pulmonary artery or one of its larger branches characteristically causes sudden severe dyspnea accompanied by a sense of substernal oppression and the rapid development of a state of shock If the patient survives the onset of the attack fever and leukocytosis usually appear within twenty four hours The clinical picture therefore is similar to that of acute myocardial infarction McGinn and White[45] pointed out that sudden occlusion of a large pulmonary artery causes prompt dilatation and failure of the chambers of the right side of the heart, and they termed this cardiac disturbance the acute cor pulmonale Several clinical details were described which serve to differentiate the condition from myocardial infarction The most important of these are an increased pulsation palpable in the second left intercostal space adjacent to the sternum, accentuation of the pulmonary second sound, the frequent occurrence of gallop rhythm over the pulmonary area, and,

unless the patient is in deep shock, engorged and distended neck veins in the absence of signs of passive congestion in the lungs There is one feature which may be a source of con fusion, namely, that in acute cor pulmonale a pericardial friction rub occasionally develops in the second and third left intercostal spaces adjacent to the sternum, apparently due to irritation of the pericardium by the dilated pulmonary artery and right ventricle The fact that this may occur means that detection of a friction rub in association with symptoms sug gestive of myocardial infarction cannot be interpreted as positive evidence that infarction is present Acute cor pul monale also causes transient changes in the electrocardio-gram, and these changes occur in approximately one third of all cases of pulmonary embolism the incidence being higher in cases with shock than in those without [46] The characteristic pattern consists of right axis deviation with a prominent wide S wave in lead I, a depressed RS T segment in lead II and a Q wave and inverted T wave in lead III The RS T segments are usually of shortened duration The picture in general resembles that of posterior myocardial infarction with right axis deviation but in acute cor pulmonale the changes usually last for several hours only while in posterior infarction they persist for a much longer time

Dissecting aneurysm of the aorta causes a clinical picture which often suggests acute myocardial infarction The onset is sudden and is characterized by severe pain in the anterior chest frequently with radiation to the back and legs and usually described by the patient as crushing or tearing in quality The pain usually lasts for forty eight hours or longer and often persists in greater or lesser degree until death occurs Repeated injections of morphine sulfate give only gradual and partial relief Fever and leukocytosis generally develop within the first twenty four hours Although there is considerable similarity to the picture of acute myocadial infarction, White, Badger, and Castleman[47] directed atten

tion to a number of features which are of value in differential diagnosis In dissecting aneurysm, the severe pain is abrupt in onset, while in myocardial infarction it develops more gradually The frequent radiation of the pain of dissecting aneurysm to the back and legs also is of diagnostic importance Particular emphasis is to be placed, however, on the maintenance of hypertension throughout the acute illness in dissecting aneurysm, the lack of diminution in the intensity of the heart sounds, and the absence of electrocardiographic signs of myocardial infarction

Complications and Sequelae The most important complications of myocardial infarction are thrombo-embolic accidents, congestive heart failure, disturbances of cardiac rhythm, rupture of the ventricle, and perforation of the interventricular septum The most frequent sequelae are angina pectoris, periarthritis of one or both shoulders with or without an associated painful disability of the hand, aneurysm of the ventricle at the site of the infarct and permanent reduction in myocardial reserve as indicated by dyspnea on limited exertion

Thrombotic or embolic accidents, including further myocardial infarction and embolism in the pulmonary or systemic circulation, complicate convalescence in approximately 35 per cent of all cases of acute myocardial infarction and usually develop within the first three weeks after the onset of the illness In Nay and Barnes[48] series of 100 consecutive cases of acute infarction there were 15 in which a second infarction occurred A second infarct may be due to extension centrally of a thrombus which caused the original attack or may involve an entirely different area of the myocardium secondary either to thrombosis of an artery in that region or to prolonged myocardial anoxia without coronary occlusion The reduction in blood pressure and the increased coagulability of the blood incident to the first infarct probably are the two most important factors which favor these occurrences

A second infarction may also result from dislodgment of an embolus from a fresh coronary thrombus with consequent obstruction of a peripheral branch of the originally involved artery This, however, is believed to be unusual

According to Blumer,[48] mural thrombi form within the ventricles in 50 per cent of all cases of myocardial infarction and approximately 14 per cent of the cases present clinical evidence of embolism arising from this source When the symptoms of acute infarction have been mild or incorrectly interpreted, the occurrence of embolism in the systemic or pulmonary circulation may first point the way to a correct diagnosis Since infarction of the left ventricle is more frequent than of the right the embolus more commonly lodges in a systemic artery The arteries of the brain extremities and spleen are predominantly affected

Pulmonary embolism is a relatively common complication of myocardial infarction but the emboli frequently arise from areas of thrombosis in the veins of the lower extremities or pelvis rather than from intramural thrombi within the right ventricle Inactivity of the extremities, reduced arterial blood pressure and increased coagulability of the blood probably are the most important factors which predispose to the development of these venous thrombi

The most common arrhythmia in acute myocardial infarction is the result of ventricular premature beats When these are few in number they are of little significance, but their occurrence at frequent intervals may be a precursor of ventricular paroxysmal tachycardia This arrhythmia, in turn, is one of the most serious complications of myocardial infarction and may be directly responsible for the development of congestive heart failure acute pulmonary edema or severe vasomotor collapse At times also it may be a forerunner of ventricular fibrillation which almost always results in sudden death Ventricular tachycardia is recognized most precisely by means of the electrocardiogram but Levine[50] has pointed

out that it often can be identified by the findings on physical examination alone The heart rate is very rapid usually between 180 and 200 beats per minute, and the rhythm, at first impression, appears to be regular With careful ausculta tion, however, an occasional fleeting irregularity usually can be detected, and this serves to distinguish the condition from auricular paroxysmal tachycardia Furthermore, if one listens carefully over the apex, an occasional accentuation of the first heart sound for a single beat frequently can be detected In ventricular paroxysmal tachycardia, the auricles and ventricles beat independently, and the accentuation of the first sound at intervals is due to simultaneous auricular and ventricular systole In auricular paroxysmal tachycardia the auricles and ventricles contract in normal sequence, and the intensity of the first heart sound therefore, does not vary As an additional point in differential diagnosis, pressure on the carotid sinus does not affect the heart rate in ventricular tachycardia, but in auricular tachycardia it often causes an abrupt return to normal sinus rhythm

Rupture of the ventricle at the site of an acute infarct occurs occasionally and is, of course, promptly fatal White and his associates[51, 52] found ten examples in 105 cases of acute myocardial infarction at the Massachusetts General Hospital and sixteen instances in 22 cases of sudden death from acute infarction in mental institutions In all but 3 of these 26 cases, rupture occurred within two weeks of the onset of the attack There was no case in which perforation oc curred through an old healed infarct It undoubtedly is very significant that in none of the cases which occurred in mental institutions had the diagnosis of myocardial infarction been made during life, and presumably therefore the patients had not been kept at strict bed rest

Perforation of an infarcted portion of the interventricular septum is an unusual complication that can be recognized accurately and is compatible at times with survival of the

patient for several years The condition is characterized by the appearance of a loud systolic murmur usually maximal in the fourth and fifth intercostal spaces to the left of the sternum and often accompanied by a palpable thrill

Of the sequelae of acute myocardial infarction angina pectoris is by far the most common Many patients who did not have angina pectoris before the infarction and most of those who had antecedent angina experience typical attacks after they have completed the period of rest in bed and have resumed a variable degree of activity Occasionally however an individual who had angina pectoris before the occurrence of myocardial infarction experiences no further paroxysms for months or years or even permanently

In approximately 15 per cent of all cases of acute myocardial infarction persistent pain in the shoulder region develops during the period of rest in bed or within the following few months [53] One or both shoulders may be affected but the left shoulder is more commonly involved than the right The pain may be mild or severe, lasts for several weeks or months and is usually increased by movement of the arm but not by walking In patients who experience attacks of angina pectoris after myocardial infarction the pain may be greatly increased during, and for some time after the anginal seizures Examination of the involved shoulder usually reveals limitation of movement, especially of abduction and external rotation with tenderness about the joint or over the lateral aspect of the upper part of the arm The common clinical picture is thus identical with that of periarthritis of the shoulder At times however pain and a sensation of weakness are the only symptoms, there is no appreciable limitation of motion, and no tenderness is present In about one third of the cases, pain, swelling, tenderness, and limitation of movement also develop in the joints of the fingers, hand, and wrist on the affected side and occasionally other joints are involved as well Although the mechanism by which

the pain in the shoulder is produced is not known, lack of movement of the shoulder during the period of strict rest and subsequently limited activity probably is an important factor The changes in the hand and wrist appear to be the result of a trophic disturbance produced in some way by vasomotor impulses arising in the painful shoulder

There are certain practical reasons why the occurrence of persistent pain in the shoulder as a sequel to myocardial in farction is deserving of emphasis When the pain first appears, many patients immediately assume that it has resulted from some further injury to the heart The physician, therefore, is consulted in alarm, and it obviously is of great importance that he be able to interpret the symptoms correctly Further more, when one is consulted by a patient because of per sistent pain in one or both shoulders the possibility that the condition may have followed unrecognized myocardial in farction should be borne in mind A careful cardiovascular history should be taken, and if suggestive symptoms have been experienced, electrocardiograms should be made

The ventricular wall at the site of myocardial infarction loses its contractile power, and, as repair progresses, the necrotic muscle is replaced by connective tissue During each systolic contraction of the heart, the non contractile tissue of the infarct is subjected to the force of the intraventricular pressure, and within a few days or weeks an appreciable out ward bulge may develop in this region Postmortem ex amination reveals the presence of a ventricular aneurysm in approximately 9 per cent of all cases of myocardial infarction in which the patient did not die soon after the onset of the attack [54] A distinction must be made, however, between anatomic and functional aneurysm A slight but definite aneurysmal bulge may be present during life while intra ventricular pressure is being exerted on the thinned tissue at the site of the infarct, and yet at necropsy no evidence of aneurysm may be found unless pressure studies are made

Master and his associates[55] were able to demonstrate systolic outward expansion of the infarcted area by fluoroscopic examination in one half of their cases Comparison of these figures with those obtained from autopsy studies indicate the approximate frequency of functional aneurysm without an anatomic sac Functional aneurysm of the ventricle can be diagnosed only by fluoroscopic examination, but anatomic aneurysm often causes a distinct bulge of the ventricular wall in the roentgenogram and may give rise to definite changes on physical examination Fulton[56] has pointed out that there may be an abrupt localized thrust or pulsation at the apex or medial to it due presumably to the systolic expansion of the aneurysm Such a finding may first suggest the diagnosis Calcium may be deposited in the wall of the aneurysm as time goes on, and this may assist in the roentgenologic demonstration of the condition It should be emphasized that ventricular aneurysm is not attended by the danger of rupture and is compatible with several years of life

Prognosis Acute myocardial infarction is always a serious condition and only a guarded prognosis is justified especially during the early part of the illness The sudden interruption of the blood supply to an area of the myocardium may cause instantaneous death due to ventricular fibrillation or abrupt cardiac arrest or death may occur several hours later during profound shock On the other hand, the patient may survive the period of pain and shock only to die of congestive heart failure rupture of the ventricle, ventricular fibrillation an embolic accident, or a second myocardial infarction during the first three weeks The first two weeks of the illness constitue the period of greatest danger and patients who survive the third week generally recover although the degree of recovery ultimately attained varies within wide limits The prognosis is better in patients less than forty years of age than it is in those belonging to older age groups and is better in men than in women Factors which affect the mortality

rate adversely include shock or pain of long duration, a pronounced and sustained fall in systolic blood pressure and pulse pressure, persistent elevation of the heart rate above 100 beats per minute, frequent ventricular premature beats, auricular fibrillation, ventricular paroxysmal tachycardia, high grade auriculoventricular block, intraventricular block, gallop rhythm pulsus alternans, more than slight enlargement of the heart, and congestive heart failure The location of the infarct has very little influence on the outlook, but the size of the infarct and the simultaneous occurrence of two or more areas of infarction have a most important effect The best available statistics concerning the over all prognosis of myocardial infarction are those of Bland and White [57] Nineteen per cent of their patients died during the first month, and 31 per cent lived for more than ten years One third of the patients who survived the first month ultimately recovered to the point of being able to carry on normal activities without symptoms Congestive heart failure was responsible for death in 75 per cent of the patients who died

Treatment The first aim in the treatment of acute myocardial infarction is to relieve the pain For this purpose either papaverine hydrochloride 30 to 90 mg ($\frac{1}{2}$ to $1\frac{1}{2}$ gr), or aminophyllin, 0 48 Gm ($7\frac{1}{2}$ gr), may be administered by slow intravenous injection Of the two preparations, papaverine seems to be the more effective, but it has not been used extensively enough as yet to assess its true value In certain patients either drug gives almost immediate relief from the pain, but if the one employed does not prove effective it should be followed promptly by the hypodermic administration of morphine sulfate The initial dose of this drug is usually 15 mg ($\frac{1}{4}$ gr), although whenever the pain is exceptionally severe, one should not hesitate to administer 30 mg ($\frac{1}{2}$ gr) Additional doses of 15 mg are given at intervals of one half hour or so if the distress continues unabated, but the total amount administered should not ex

ceed 60 mg (1 gr) and the drug should not be employed after the pain begins to subside Morphine causes nausea in many patients and induces vomiting in some and the exertion of vomiting is, of course, undesirable in acute myocardial infarction.

Immediately after the administration of papaverine or aminophyllin, and regardless of whether morphine is necessary or not the patient should be given atropine sulfate by subcutaneous injection LeRoy and Snider[38] have demonstrated that the occurrence of ventricular fibrillation after ligation of a coronary artery in dogs is due to generalized myocardial anoxemia caused by vagal coronary constriction If the vagal vasoconstriction is reduced or abolished by adequate amounts of atropine, the incidence of sudden death after coronary artery ligation is considerably reduced In man the usual initial dose is 0 8 mg (1/75 gr), and additional doses of 0 4 mg (1/150 gr) are given every eight hours during the first three or four days

The patient should be placed in bed in whatever position is most comfortable for him as soon as possible after the onset of symptoms and should not be disturbed by too frequent examinations Because of the shock and profuse perspiration which often are present the body should be kept warm and as soon as the patient is more comfortable and is free from nausea and vomiting, small amounts of fluids should be offered at frequent intervals In states of severe shock in which the systolic blood pressure falls to 80 mm of mercury or less the slow intravenous administration of small amounts of plasma, 200 or 250 cc. has been recommended[21] The patient must be observed closely during the injection, and if any evidence of further embarrassment of the circulation develops, the plasma must be discontinued immediately The injection may be repeated at intervals of two to four hours until the systolic pressure rises to and remains above 100 mm The use of plasma in the treatment of shock prob-

ably is to be preferred to the administration of stimulants such as caffeine with sodium benzoate

In the more severe attacks of myocardial infarction which are accompanied by prolonged pain, cyanosis, and intense dyspnea, oxygen should be given as promptly as possible, preferably by means of an oxygen tent This measure not only reduces the cyanosis and dyspnea but may also lessen the intensity and shorten the duration of the pain

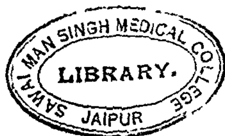
The most promising recent addition to the treatment of myocardial infarction consists of the use of the anticoagulant agent dicumarol The drug has not been administered in a sufficient number of cases to establish its value positively, but its use is logical and the results reported thus far have been encouraging [59 60 61] Thrombo embolic accidents of various kinds, as has been pointed out, constitute one of the most important complications of myocardial infarction, and any measure which offers a reasonable prospect of reducing their frequency deserves a thorough clinical trial Dicumarol prolongs the prothrombin clotting time of the blood, and this should prevent or at least reduce the frequency of, thrombus formation in all parts of the vascular system As a result, the incidence of mural thrombi in the ventricles should be lessened there should be less tendency to propagation centrally of a thrombus in a coronary artery or the formation of additional thrombi in other coronary branches, and the development of thrombi in peripheral veins should be prevented The frequency of embolic accidents and the incidence of a second myocardial infarction during the first few weeks after the initial attack therefore should be greatly reduced, and this, in turn, should result in considerable improvement in the immediate prognosis of the illness

Dicumarol should not be employed if facilities are not available for measuring the prothrombin time of the blood and should not be given to patients who have renal or hepatic insufficiency or a blood dyscrasia with hemorrhagic tend

encies The prothrombin time is measured before the first dose of the drug is administered again on the following morning, and subsequently either daily or every other day Dicumarol requires from twenty four to forty-eight hours to attain its maximum effect, and until the desired lengthening of the prothrombin time has been attained it may be best to also administer heparin by intravenous injection in doses of 50 mg every 4 hours The amount of dicumarol to be administered in one day is given as a single dose On the first day 300 mg are given, and on the second day 200 mg Subsequent dosage must be individualized for each patient The aim in treatment is to prolong the prothrombin time to a prothrombin concentration between 20 per cent and 30 per cent of normal (method of Quick) In many patients this can be accomplished by giving 200 mg every forty-eight hours after the second dose Treatment is continued for three weeks and is then discontinued gradually If at any time the prothrombin time becomes unduly prolonged and hemorrhagic manifestations occur menadione bisulfite (synthetic vitamin K) is given by intravenous injection in doses of 72 mg once or twice a day until the condition is corrected Small transfusions of fresh whole blood may be given also if serious bleeding occurs

After the pain and initial shock of acute myocardial infarction have been controlled, most patients require little medication other than dicumarol Atropine is continued in the prescribed dosage for three or four days, and it probably is best to administer adequate amounts of one of the purine derivatives such as theobromine and sodium acetate in doses of 0 5 gm (7½ gr) four times a day For the first week or so a mild sedative, such as phenobarbital frequently is advisable either in small divided doses during the day or at bedtime to control restlessness Digitalis is administered only to patients in whom evidence of congestive heart failure develops or in the event of auricular fibrillation with a rapid ventricu

one half hour each day A few steps about the room are allowed at the end of the first week, and as the time up is lengthened each day, the amount of walking is gradually increased When the time up reaches four hours twice a day, a schedule is adopted which permits the patient to get up leisurely in the morning, stay up until after lunch, and then after a two hour rest period, remain up until an early retiring hour Resumption of business or professional activities is not allowed for three to six months, depending on the severity of the attack





Chapter IV

ACUTE CORONARY FAILURE

IT HAS been recognized for some time that certain patients who have coronary artery disease experience attacks of substernal pain of longer duration than the pain of angina pectoris but do not subsequently present clinical or electro cardiographic evidence of myocardial infarction The syn drome received very little notice, however, until the recent studies of Blumgart and Schlesinger[2, 35] sharply focused atten tion on it Blumgart and Schlesinger pointed out that the physiologic basis for the pain in this condition is the same as for the pain of angina pectoris namely a reversible myo cardial anoxia, but the anoxia lasts longer than in angina pectoris and yet not long enough to cause myocardial necro sis The term "coronary failure" was applied to the syndrome, but a title that would more clearly separate it from angina pectoris and myocardial infarction is desirable Recognition of the condition is a matter of practical importance for, among other considerations the attacks at times prove to be the forerunner of acute myocardial infarction

Pathology In certain instances, acute coronary failure is due to the presence of extensive coronary artery disease without fresh occlusion of a vessel This probably is the case in those patients in whom the seizures are precipitated by a reduction in coronary blood flow due to paroxysmal disturb ances of the heart rhythm or hypotensive states Other at tacks are attributable to partial or complete acute occlusion of a coronary branch without myocardial infarction Infarc tion does not occur in these cases because coronary artery disease of sufficient degree has been present for a sufficient length of time to result in the development of adequate

pathways of collateral circulation Obstruction of a coronary branch therefore does not interfere with the nutrition of the myocardium to the extent necessary to cause necrosis of tissue, although it does cause a prolonged period of anoxia Blumgart and Schlesinger believe that when coronary failure occurs as the result of circumstances which, when previously experienced, failed to provoke an attack, the symptoms probably are due to acute coronary occlusion without myocardial infarction This would apply to paroxysms which are induced by emotion or exertion that formerly had caused no symptoms or had induced only typical seizures of angina pectoris It probably is the mechanism also of attacks which occur in the absence of apparent precipitating factors

Symptoms and Signs Paroxysms of acute coronary failure may occur while the patient is at rest or they may be induced by any factor which increases the demand on the heart for work or decreases coronary blood flow Exertion, emotion, exposure to cold, overeating paroxysmal auricular fibrillation or flutter, paroxysmal tachycardia, and hypotension due to shock or hemorrhage are responsible for many of the seizures When paroxysmal tachycardia or a paroxysmal arrhythmia causes an attack, the pain usually lasts as long as the heart rate remains elevated

The pain in acute coronary failure is similar to that of angina pectoris but usually lasts for at least twenty minutes and often much longer It frequently is very severe, often is accompanied by some respiratory distress and sweating and may be attended by a transient rise in the arterial blood pressure Nitroglycerine usually fails to give more than partial and temporary relief, and the paroxysm often is followed by a feeling of profound weakness Fever, leukocytosis, and an elevated sedimentation rate do not occur, and there is no subsequent progressive decline in the blood pressure

Electrocardiograms made during an attack may or may not show changes similar to those which occur in many pa

tients during seizures of angina pectoris. As in the case of angina pectoris the changes are of short duration, and the record promptly returns to its original state when the pain subsides. Subsequent tracings fail to show the serial changes of acute myocardial infarction.

Treatment. The treatment of acute coronary failure consists of measures to control the pain and, if possible, correct the precipitating factors; a period of rest in bed; and the administration of dicumarol as a prophylactic measure against the possible development of a coronary thrombus or propagation of a thrombus that already has formed. When the patient is seen while the pain is still present, papaverine hydrochloride or aminophyllin are given by intravenous injection in the same amounts as in the treatment of acute myocardial infarction. If the preparation employed is not effective, it should be followed promptly by a hypodermic injection of morphine sulfate, 15 mg ($\frac{1}{4}$ gr), and atropine sulfate, 0.8 mg ($\frac{1}{75}$ gr).

If ventricular paroxysmal tachycardia is present, quinidine sulfate should be administered in the manner outlined previously for this arrhythmia. Patients who have paroxysmal auricular fibrillation or flutter are digitalized rapidly by the use of digitoxin and are given quinidine in the same amounts as employed in the treatment of ventricular tachycardia. Rapid digitalization is also indicated when auricular paroxysmal tachycardia is present and does not respond to carotid sinus or ocular pressure and breath holding. If hemorrhage or shock have been responsible for the attack, small transfusions of whole blood or plasma should be given slowly.

Because acute coronary failure may be the precursor of acute myocardial infarction, the demands on the heart muscle should be reduced to a minimum as quickly as possible. Rest in bed is necessary and should be continued for ten to fourteen days. Dicumarol is administered during this time in the

same manner as in the treatment of acute myocardial infarction All patients are placed on one of the xanthine preparations (theobromine and sodium acetate, theobromine and calcium salicylate, or aminophyllin) for a period of at least a few months

Chapter V

PAROXYSMAL CARDIAC DYSPNEA (CARDIAC ASTHMA)

THE TERM paroxysmal cardiac dyspnea is applied to certain attacks of severe shortness of breath which occur in patients who have serious organic heart disease The initial seizure occasionally gives the first warning of a damaged heart, but more often the patient has experienced dyspnea or substernal pain on effort for some time previously The paroxysms are frequently accompanied by asthmatic breathing, and because of this the condition often is referred to as cardiac asthma Many attacks, however and especially those of lesser severity are not attended by wheezing or asthmatic râles It seems desirable, therefore, to use the term paroxysmal cardiac dyspnea for the entire group of cases and designate as cardiac asthma only that subgroup in which asthmatic breathing is present

Etiology and Pathogenesis In all but a few cases paroxysmal cardiac dyspnea is due to acute failure of a left ventricle that has been damaged previously by coronary artery disease, hypertension or aortic valve disease Coronary artery disease with or without hypertension is responsible for approximately two thirds of the cases In a small group of patients, the attacks result from the presence of well marked mitral stenosis without myocardial failure A series of such cases has been reported by McGinn and White[63] who point out that, in contrast to paroxysms due to acute left ventricular failure, the attacks in these patients usually are precipitated by exertion, emotional upsets or paroxysmal tachycardia When the heart rate is accelerated by any of these factors the hypertrophied right ventricle expels blood into the pul

monary vessels more rapidly than it can pass through the narrowed mitral orifice The resulting acute pulmonary hypertension and congestion cause a paroxysm of severe dyspnea which is indistinguishable from that of the more common type of attack The more common form of seizure, however, results from an entirely different mechanism and usually occurs after the patient has been at rest in the recumbent position for a few hours

When a patient suffering from impaired myocardial reserve assumes the recumbent position, certain factors become operative to bring about an increasing degree of pulmonary congestion If edema is present, a redistribution of the edema fluid occurs because of the effect of gravity,[64] a part of the fluid shifts from the legs to the lower back and lungs, and pulmonary râles may develop for the first time In addition the venous pressure in the lower portion of the body diminishes and this change would naturally be expected to result in absorption into the blood stream of a certain part of the edema fluid in the lower extremities That this actually occurs has been demonstrated by Perera and Berliner[65] who found that a considerable decrease in serum protein concentration and an increase in plasma volume develop in the recumbent position If the right and left ventricle are equally affected by myocardial disease, the increase in blood volume will be divided between the systemic and pulmonary circulation in the normal ratio If predominant weakness of the left ventricle is present, however, there will be a gradual accumulation of a disproportionate amount of the blood in the pulmonary vessels As a result the vital capacity of the lungs, which is already diminished by the assumption of the recumbent position and frequently by pre existing pulmonary congestion, is still further reduced as the degree of pulmonary engorgement increases Within a few hours after the patient lies down a stage is reached where all that is necessary to initiate the attack of paroxysmal dyspnea is

some factor which acts as a trigger mechanism This is supplied by anything that wakens the patient and most commonly by cough due to the accumulation of secretions in the respiratory tract, Cheyne Stokes respiration noise, disturbing dreams, and distention of the urinary bladder The patient wakens in acute respiratory distress and all of the clinical features of the seizure rapidly follow If the attack is severe or of long duration, acute pulmonary edema may develop

Harrison and his co workers[65] have made extensive studies of the manner in which the factors that commonly precipitate attacks of paroxysmal cardiac dyspnea exert their effect They demonstrated that, in patients with myocardial insufficiency, coughing causes a great increase in the respiratory minute volume and induces respiratory distress In one of their patients, continuous coughing for two minutes precipitated a typical attack of paroxysmal dyspnea complicated by acute pulmonary edema They also pointed out that forced breathing usually causes coughing in individuals who have passive congestion of the lungs The respiratory distress of paroxysmal cardiac dyspnea therefore will tend to induce still further coughing which in turn makes the dyspnea worse A vicious cycle is thus completed Harrison also directed attention to the fact that at the beginning of each inspiration following cough there is a sudden inflow of blood into the right auricle and a consequent rise in the pressure in that chamber This rise in pressure causes reflex stimulation of the respiratory center and a further increase in the degree of dyspnea

Noise, distressing dreams, and distention of the urinary bladder cause a rise in respiratory minute volume just as does cough They will therefore induce dyspnea in patients who have pulmonary congestion and a reduced vital capacity and will contribute to the development of an attack of paroxysmal dyspnea in the same manner as does cough It must be

emphasized, however, that in order for any of the factors to precipitate a seizure, the condition of the heart must be such that failure of the left ventricle predominates over any accompanying weakness of the right ventricle In other words, the situation must be one that favors the development of progressive pulmonary congestion

When a patient wakens in an attack of paroxysmal cardiac dyspnea, he automatically makes a number of adjustments which frequently give prompt relief from the symptoms A sitting or standing position is assumed, and the factor which acted as the trigger mechanism is eliminated as quickly as possible, either by the expectoration of mucus to relieve the cough, or by recovery from the fright of a noise or distressing dream, or by emptying the bladder The vital capacity of the lungs is greater in the sitting or standing position than in the recumbent posture in all individuals When pulmonary congestion is present, the beneficial effect of the upright position is enhanced by pooling of blood in the veins of the lower part of the body and a consequent decrease in the engorgement of the pulmonary vessels In the sitting posture also, fluid escapes from the blood into the tissues of the lower part of the body and the blood volume diminishes [47] This, in turn, directly reduces the load upon the heart There is evidence, furthermore, that the minute volume output of the heart is decreased in the upright position [48] The combined effect of these corrective factors may give prompt relief from the attack of paroxysmal dyspnea and enable the patient to return to bed within a few minutes

The reason why asthmatic breathing occurs in some patients during attacks of paroxysmal cardiac dyspnea and not in others is not known The difference, in some instances, probably is due to differences in the severity of the attack One wonders, however, whether the occurrence of typical cardiac asthma may not depend on the presence of a constitutional factor which predisposes the patient to broncho

spasm in response to the rapid development of pulmonary congestion This factor would be comparable to the factor which determines the occurrence of bronchoconstriction in allergic individuals in response to an offending allergen It is to be emphasized, however, that patients who have cardiac asthma are not merely individuals with bronchial asthma in whom left ventricular failure has developed

Symptoms and Signs Although, in an occasional case attacks of paroxysmal cardiac dyspnea are precipitated by exertion, the seizures usually occur at night and begin after the patient has been asleep for a few hours Typically, the patient retires in a comfortable state and then wakens about one or two o'clock in the morning in great respiratory distress Frequent coughing and a sense of suffocation are present, and the individual is forced to sit up or stand in order to breathe In mild attacks a small amount of sputum is raised within a few minutes, and with this the cough ceases and the dyspnea subsides The patient returns to bed, and the remainder of the night usually passes without incident In severe paroxysms, on the other hand the dyspnea and cough rapidly become worse, and asthmatic breathing with both inspiratory and expiratory difficulty frequently develops The patient may rush to an open window and support himself with his hands on the sill in an attempt to obtain more air The attacks last from several minutes to a few hours, and if they are of sufficient severity acute pulmonary edema may supervene and large amounts of frothy, blood tinged sputum may be expectorated Although death may occur during a seizure, the symptoms usually subside gradually or are controlled by treatment, and the patient then remains comfortable for the rest of the night Further attacks may occur only at infrequent intervals or they may develop night after night and with great regularity as to the time of onset When frequent seizures are experienced, the victim soon comes to dread going to bed and may discover that the

paroxysms can be avoided or at least diminished in severity by sleeping in a chair

Examination during an attack reveals an orthopneic, apprehensive, and often somewhat cyanotic individual Usually there is frequent coughing, often severe and paroxysmal in nature The peripheral venous pressure is elevated in many cases, and the jugular veins may be engorged to the angle of the jaw even with the patient in the sitting position The thorax becomes more or less fixed in the inspiratory position, and respiration is largely diaphragmatic Moist râles are present over the base of the lungs, and if acute pulmonary edema develops the râles increase greatly in number and become coarse and bubbling in character In cases of cardiac asthma, sibilant and sonorous rales appear over all lung fields Auscultation of the heart reveals accentuation of the pulmonary second sound and frequently a gallop rhythm The heart rate is accelerated, and the arterial blood pressure is almost always elevated

Differential Diagnosis When paroxysmal cardiac dyspnea is accompanied by the development of pulmonary rhonchi and asthmatic breathing, the condition must be differentiated *from bronchial asthma Usually this is not difficult*, for in cardiac asthma examination of the chest reveals asthmatic râles only during the attack In bronchial asthma, on the other hand, at least a few rhonchi persist for a period after the attack, and often a considerable number are to be heard at all times during the intervals between paroxysms In uncomplicated bronchial asthma, furthermore, moist râles are not present over the base of the lungs, while râles of this type invariably accompany the attack of cardiac asthma and usually persist in smaller numbers after the paroxysm has ended The response to epinephrine cannot be used as a guide in differential diagnosis because the drug may give just as prompt relief from the attack of cardiac asthma as from

bronchial asthma, although its use in cardiac asthma is not to be recommended

Congestive heart failure may develop in any patient who has bronchial asthma or emphysema of the lungs and some form of organic heart disease The onset of failure in such patients usually is accompanied not only by increasing dyspnea and orthopnea but also by an increase in the severity of the asthmatic breathing and in the number of rhonchi This is not cardiac asthma and the features which serve to distinguish it from cardiac asthma are a history of asthma in the past, the evidence of emphysema on physical examination, the persistent rather than periodic presence of rhonchi and the occurrence of asthmatic attacks without relation to a preceding period of recumbency Symptoms and signs of asthma are constantly present during the period of congestive failure but frequently improve and may even disappear as the heart becomes compensated In contrast to this patients with cardiac asthma suffer from asthmatic breathing only during the paroxysms of dyspnea

Paroxysmal cardiac dyspnea must also be differentiated from paroxysmal dyspnea due to Cheyne Stokes respiration Cheyne Stokes respiration may act at times as the factor which precipitates an attack of paroxysmal cardiac dyspnea but the two conditions otherwise are unrelated In Cheyne Stokes respiration the breathing is periodic and causes dyspnea at the beginning of sleep The patient often wakens repeatedly with shortness of breath at the end of the periods of apnea In paroxysmal cardiac dypsnea, on the other hand the breathing is regular and the attacks occur after the patient has been asleep for two or more hours

Prognosis Paroxysmal cardiac dyspnea due to failure of the left ventricle indicates the presence of serious organic heart disease and implies a grave prognosis The persistence of gallop rhythm, pulsus alternans or pulmonary congestion

during the intervals between attacks increases the seriousness of the outlook The average duration of life after the first paroxysm is less than two years, and approximately one half of the patients die within one year after the first seizure[69] The most common causes of death are congestive heart failure and acute myocardial infarction

Treatment The treatment of paroxysmal cardiac dyspnea consists of certain measures to terminate the attacks and others to prevent their recurrence The most effective measures in the treatment of a paroxysm are the administration of morphine sulfate and aminophyllin, and the assumption of the upright position Morphine exerts its beneficial effect by depressing the respiratory and higher cerebral centers Dyspnea is therefore lessened, and the patient's apprehension and anxiety are reduced The drug should be administered hypodermically as early in the attack as possible, usually in a dose of 15 mg (¼ gr), and if the symptoms are of more than mild degree it should be followed promptly by the intravenous injection of aminophyllin, 0 48 Gm (7½ gr) in 20 cc of solution Aminophyllin may bring about prompt and lasting improvement Its value has been attributed in the past principally to its effect on the coronary circulation, but its action on the bronchial musculature probably is of equal importance Aminophyllin has the power to relieve bronchial constriction which is known to be present during the paroxysm of cardiac asthma and must be responsible for a considerable part of the dyspnea If its administration does not prove sufficiently helpful, a second injection of morphine should be given twenty minutes or so after the first

Morphine, aminophyllin, and the upright position may fail to give relief at times, and other measures must then be employed Nitroglycerine may be effective, especially in patients with hypertension The administration of oxygen by means of a tent, B L-B mask, or nasal catheter is of great

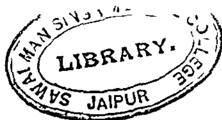
value and should be instituted promptly when facilities are available When the venous pressure is elevated venesection should be performed, with the removal of 250 to 500 cc of blood This reduces the circulating blood volume and as a result the work of the heart is lessened and pulmonary congestion diminished An effect similar to that of venesection can be obtained by applying blood pressure cuffs to the four extremities and inflating them to a pressure just above the diastolic blood pressure [70]

When paroxysmal cardiac dyspnea progresses to acute pulmonary edema ouabain or a suitable preparation of digitalis should be given in proper amounts by intravenous injection It is essential, of course, to ascertain beforehand that the patient has not received digitalis during the preceding two weeks

The most helpful measures for preventing recurrent seizures are limitation of physical activity the administration of digitalis, a diet low in sodium, and the use of diuretic drugs All patients who have experienced an attack of paroxysmal dyspnea due to left ventricular failure should be completely digitalized and should receive subsequent daily maintenance amounts of the drug Digitalis alone may suffice to prevent further seizures in those who have had only mild attacks All patients and especially those who have clinical edema, should be placed on a diet containing not more than 2 or 3 grams of sodium chloride per twenty four hour ration A low sodium intake is of established value in preventing the accumulation of edema fluid and in hastening its elimination when already present When edema is present, one of the mercurial diuretics should be given by intravenous or intramuscular injection daily or every other day until its administration no longer causes diuresis Ammonium chloride may also be prescribed in doses of 6 to 12 grams daily by mouth If, in spite of these measures, paroxysms continue to recur

each night, they frequently can be prevented by the administration of aminophyllin in the evening either by intravenous injection or in the form of a rectal suppository

When attacks of paroxysmal cardiac dyspnea are mild and of short duration and there are no other manifestations of congestive heart failure, the patient usually can be allowed to continue his daily activities but at a reduced pace For individuals who have experienced more severe seizures or who present signs of congestive failure, a period of strict rest in bed is advisable Since the occurrence of attacks is favored by the recumbent position all patients should be instructed to sleep well propped up in bed If there have been severe paroxysms and a period of strict rest is to be carried out, it often is best to elevate the head of the bed on "shock blocks" for a few days until digitalis diuretic drugs, and the reduced intake of sodium have improved the condition of the heart and eliminated whatever edema was present Sedatives should be used with caution since their depressant effect upon the respiratory center may favor rather than hinder the development of attacks



Chapter VI

HEART BLOCK AND DISTURBANCES OF CARDIAC RHYTHM

Heart Block Since normal function of the conduction system of the heart depends upon the integrity of the blood supply to the specialized conducting tissue it is not surprising that coronary artery disease should be a common cause of auriculoventricular and intraventricular (bundle branch) block In individuals beyond the age of fifty years coronary arteriosclerosis is responsible for the great majority of cases of both of these types of block All degrees of either type may result from minimal but appropriately placed coronary changes but on the other hand, advanced coronary disease with extensive fibrosis in the conducting tissues may fail to cause a conduction disturbance of any kind There is therefore, no parallelism between the degree of coronary arteriosclerosis and the presence of auriculoventricular or intraventricular block There is also no direct relation between the presence of either form of block and the life expectancy of the patient Individuals who present one of these conduction disturbances as the only evidence of coronary disease and who have no other abnormal cardiovascular findings such as hypertension or enlargement of the heart may live for years without the least inconvenience and may die eventually of causes quite unrelated to the heart On the other hand, the occurrence of either type of block in patients who have angina pectoris myocardial infarction, paroxysmal cardiac dyspnea, or congestive heart failure definitely increases the gravity of the outlook

Auriculoventricular block secondary to coronary arteriosclerosis may be of any degree from simple lengthening of

auriculoventricular conduction time to complete dissociation of the auricles and ventricles and may or may not be associated with one or more of the other manifestations of coronary artery disease In some cases the degree of block varies from time to time as the result of fluctuations in vagus tone, the effect of drugs, or changes in coronary blood flow Prolonged conduction between the auricles and ventricles, termed first degree auriculoventricular block, can be recognized with precision only by means of the electrocardiogram The higher grades, namely second degree block with occasional or regularly recurring dropped beats and complete auriculoventricular dissociation often can be diagnosed, or at least suspected from the findings on auscultation of the heart

First degree auriculoventricular block causes no symptoms, and this is true also for second degree block except in the occasional patient who experiences palpitation as a result of the dropped ventricular beats There is no effective treatment for block of these degrees, although occasionally, in the presence of high vagus tone, the administration of atropine may shorten the auriculoventricular conduction time or reduce the frequency of the dropped beats When myocardial insufficiency is present, neither first nor second degree block contraindicates the administration of digitalis Digitalization should be accomplished gradually, and the effect of the drug should be observed by daily electrocardiograms If the degree of block increases, it may be advisable to be satisfied with less than complete digitalization In favorable cases, the drug not only relieves the symptoms and signs of myocardial failure but at times also reduces the degree of block or abolishes it entirely

The higher grades of auriculoventricular block, and particularly complete auriculoventricular dissociation, may be complicated by the Adams Stokes syndrome due either to sudden marked slowing of the ventricular rate or to temporary standstill of the ventricles The attacks are characterized

by faintness, dizziness, syncope, or convulsions depending on the new heart rate or the duration of ventricular asystole Adams Stokes seizures are rare but individuals in whom they occur are liable to have repeated attacks The actual seizures usually are of such short duration that they do not require treatment, but in rare instances the ventricular stand still is of such duration that life is threatened and the intra cardiac injection of epinephrine hydrochloride 0 5 to 1 0 cc of 1 1000 solution, is indicated The most effective drugs for preventing recurrent attacks are epinephrine hydro chloride 0 3 to 1 0 cc of the 1 1000 solution subcutaneously or intramuscularly every two to four hours, ephedrine sulfate by mouth in doses of 24 to 30 mg (3/8 to 1/2 gr) every four to six hours and paredrine by mouth in doses of 40 to 60 mg (2/3 to 1 gr) three times a day

Coronary artery disease is responsible for all but a few cases of intraventricular block The block may be of the complete or incomplete, right or left bundle branch type, or of a lesser degree to which the term defective intraventricular conduction is applied The diagnosis of any form can be made only by means of the electrocardiogram, and diagnostic criteria will be found in all texts on electrocardiography None of the forms gives rise to symptoms and while each represents an important abnormality none can be credited with independent prognostic significance However when any type of intraventricular block is present in patients who have symptoms due to coronary artery disease the block is usually due to extensive rather than localized coronary changes and the prognosis therefore is serious

Premature Beats All of the recognized disturbances of cardiac rhythm may occur in patients who have coronary artery disease but it does not follow that the arrhythmia always is a direct result of the coronary changes Ventricular and other forms of premature beats, for example are fre quently ob erved in patients who have coronary heart dis-

ease, but because they also are encountered in many normal individuals no particular significance can be attached to them in the usual case Occasionally, their sudden appearance in large numbers in a patient beyond the age of 50 years may be the first evidence of coronary disease and myocardial strain In a situation of this kind, moderate limitation of physical activity and the cautious administration of digitalis are indicated and, in certain cases, will result in prompt subsidence of the irregularity The significance and treatment of frequent ventricular premature beats occurring during the early period of acute myocardial infarction has already been mentioned

Auricular Fibrillation Coronary disease ranks next to rheumatic heart disease as a cause of auricular fibrillation of both the continuous and paroxysmal forms The presence of either type of the arrhythmia in a person more than fifty years of age with no other cardiac abnormality is presumptive evidence of coronary artery disease Auricular fibrillation may occur at any age as a purely functional disturbance, but after the age of fifty the number of individuals who can be classified in this group with certainty is very small In this connection, an electrocardiogram which is normal in every respect except for the arrhythmia does not exclude coronary disease The decision as to whether a given case is of functional origin or due to coronary changes may not be possible until the clinical course has been followed for a year or more or until other evidence of coronary disease develops It must also be kept in mind that auricular fibrillation may be due to mild or so called masked hyperthyroidism, and the possibility of this condition should be considered in every case of the arrhythmia, even though evidence of obvious organic heart disease also is present

Not only may auricular fibrillation be the only manifestation of coronary heart disease, but if the ventricular rate is slow, it may cause no symptoms Usually, however, the ven

tricular rate is rapid, and the patient will have noted palpitation either at rest or on exertion Heart consciousness of this kind, accompanied at times by weakness or light headedness is especially liable to occur at the onset of the arrhythmia and is often particularly annoying in the paroxysmal form of the disturbance

In a majority of the cases in which auricular fibrillation occurs as a complication of coronary artery disease evidence of myocardial insufficiency is also present Frequently the symptoms of diminished myocardial reserve antedate the arrhythmia by months or even years, and at times auricular fibrillation does not develop until after the onset of congestive heart failure In the latter cases the abnormal rhythm appears to be more directly a result of the myocardial failure than of the underlying coronary disease By way of contrast, there is a group of patients who have few or no cardiac complaints until the development of auricular fibrillation precipitates the rapid appearance of symptoms and signs of congestive heart failure

Auricular fibrillation as has been mentioned is a not infrequent complication of acute myocardial infarction and when it occurs as such it is usually, but not always of the paroxysmal variety The arrhythmia is encountered only rarely in patients who have angina pectoris and when it does develop the attacks of angina frequently cease to recur This is probably due principally to the fact that the accompanying dyspnea and palpitation force the individual to keep his activity below the point at which the substernal distress appears

In patients who have rheumatic heart disease, one of the most serious complications of auricular fibrillation is the occurrence of embolic phenomena Accidents of this kind are much less common in auricular fibrillation due to coronary artery disease although they do occur The difference in incidence is attributable to the fact that in the rheumatic

heart, the arrhythmia almost always occurs in association with mitral valve disease, and the mitral lesion favors more marked stagnation of blood in the left auricle than occurs in auricular fibrillation with normal mitral valves Mural thrombi therefore develop in the left auricle or auricular appendage more frequently in patients with mitral stenosis, and a portion of a thrombus may become detached at any time to form an embolus

As in auricular fibrillation associated with other forms of heart disease, the two drugs employed in the treatment of cases due to coronary artery disease are digitalis and quinidine sulfate Digitalis can be used in all cases without hesitation but quinidine should be administered only to carefully selected patients and then with the expectation of a successful result in perhaps not more than 50 per cent of the group Digitalis is given, of course, with the object of reducing the ventricular rate to 70 beats per minute or slightly less and then maintaining this rate Quinidine, on the other hand, is employed to terminate the arrhythmia and re-establish normal rhythm

When auricular fibrillation due to coronary artery disease is known to be of recent origin and is not associated with congestive failure or more than slight enlargement of the heart quinidine sulfate should be administered Its use, however, should be preceded by complete digitalization of the patient, and maintenance amounts of digitalis should be given throughout the period of therapy The patient should be kept under close observation while quinidine is being administered and should remain in bed each day until at least two hours after the last dose of the drug The best results are obtained when the drug is given with an interval of two hours between doses On the first day two doses of 0 2 Gm (3 gr) are administered as a test amount to detect possible hypersensitivity If no symptoms develop, five doses of 0 4 Gm (6 gr) are given on each of the following

days, but if auricular fibrillation is still present at the end of five days, the drug is discontinued In such cases daily administration of maintenance amounts of digitalis is continued indefinitely

Quinidine is not employed if auricular fibrillation has been present for a long time or if the heart is considerably enlarged nor is it used when the arrhythmia complicates the usual case of congestive heart failure In these situations the ventricular rate should be controlled by adequate amounts of digitalis, and no attempt should be made to re establish normal heart rhythm An exception to this rule is made in rare cases of myocardial failure If, in congestive failure with auricular fibrillation the ventricular rate remains elevated and the patient does not improve in spite of all the usual measures of treatment including digitalis to the limit of tolerance, quinidine may be given [71] When the result is successful and sinus rhythm is restored the heart rate may be considerably slower than it was while auricular fibrillation was present, and this may lead to clinical improvement Treatment in this manner is attended by the danger of embolism at the time of transition to normal rhythm or shortly thereafter, but the risk is justifiable when all other measures have failed to improve the patient

In the treatment of paroxysmal auricular fibrillation due to coronary artery disease, quinidine is employed for the purpose of terminating the attacks and preventing or at least reducing the frequency of further paroxysms For the treatment of the attacks the schedule of dosage outlined above is followed, although if the patient has received quinidine before, the test doses can be omitted When the drug is used as a prophylactic agent against further recurrences, it usually is given in doses of 0 2 to 0 4 Gm (3 to 6 gr) every six or eight hours The results are frequently disappointing and the patient often can be kept much more comfortable by complete digitalization and subsequent maintenance of the

digitalized state This seldom reduces the frequency of the attacks but it does prevent the excessive rise in the ventricular rate which occurs in undigitalized patients and which is responsible for the unpleasant symptoms of a paroxysm

Ventricular Paroxysmal Tachycardia Ventricular paroxysmal tachycardia is a rare condition which is usually due to serious organic heart disease It occurs most commonly as a complication of acute myocardial infarction, and its importance, diagnosis, and treatment in this connection have been discussed earlier

Chapter VII

CONGESTIVE HEART FAILURE

MENTION has been made of the fact that acute myocardial infarction may be followed by the rapid development of congestive heart failure and that decompensation of more gradual onset may occur in patients who have angina pectoris Coronary artery disease is often responsible also for the gradual appearance of myocardial failure in individuals who have had no earlier symptoms referable to the heart Failure occurs whenever the anatomic and functional changes due to coronary disease so impair the efficiency and adaptive capacity of the myocardium that an adequate blood flow can no longer be maintained in the lungs and peripheral circulation That the shock and the loss of contractile power of an area of the ventricular muscle which result from acute myocardial infarction should cause cardiac decompensation of rapid onset is readily understandable In patients who have had angina pectoris prior to the onset of myocardial failure or in whom failure occurs without previous manifestations of coronary disease myocardial fibrosis of variable degree usually is present Symptoms and signs of myocardial weakness may be induced in these individuals by any factor which suddenly or persistently increases the work of the heart or reduces the coronary blood supply Severe exertion emotional upsets systemic infection debilitating disease the onset of auricular fibrillation or flutter, paroxysmal tachycardia thyrotoxicosis, hemorrhage, and shock are among the most important of these factors

The manifestations of congestive heart failure secondary to coronary artery disease do not differ from those of failure due to other forms of heart disease and need not be reviewed

here Treatment likewise is the same as for myocardial failure resulting from other types of cardiac disease and consists principally of the proper use of digitalis, the administration of diuretic drugs, strict limitation of the sodium content of the diet without restriction of the intake of water, sedatives as required and an adequate period of absolute rest in bed Venesection and the mechanical removal of fluid from the serous cavities of the body are required in certain patients

Levine[64] has recently emphasized that certain harmful effects may follow the sudden enforcement of recumbency in patients who have congestive heart failure These effects are to be regarded as complications against which suitable precautions should be taken, and the possibility of their occurrence does not detract from the importance of rest in heart failure The assumption of the recumbent position results in a shift of edema fluid from the lower to the upper portions of the body, facilitates the venous return to the heart, and is accompanied by an increase in the circulating blood volume, and unless corrective measures are taken, these changes may cause a decided increase in the degree of failure When moderate or severe decompensation is present, the patient should be digitalized rapidly and should receive one of the mercurial diuretics at the very beginning of treatment If the situation is urgent and there is no anemia, venesection also should be carried out with the removal of 500 to 600 cc of blood When considerable edema is present, the use of "shock blocks" under the head of the bed is to be preferred to maintaining a propped up position by elevation of the head rest alone Occasionally it may even be advisable to allow the patient to spend the first few days of treatment *in* a comfortable chair with the feet dependent

Levine also pointed out that the sitting position in bed causes increased pressure on the femoral veins and favors venous stasis in the lower extremities This predisposes to the development of phlebothrombosis which may in turn

become a source of pulmonary emboli The danger of such a complication can be reduced by avoidance of the sitting position during the early days of treatment massage and passive exercise of the lower extremities and early active movements of the legs The sitting position can be avoided by substituting the use of 'shock blocks," in the manner mentioned above, for elevation of the head rest of the bed

Every patient who has congestive heart failure of more than slight degree should have at least six weeks rest in bed If there is great difficulty in the use of the bed pan and the patient is improving satisfactorily the program may be modified after ten days or two weeks by allowing the use of a commode or a daily trip to the bathroom, but no other exception should be made After completion of the period of rest gradually increasing activity is permitted with care to avoid dyspnea and fatigue and careful observation for any return of edema Digitalis should be continued permanently in maintenance doses, and a diet low in salt content should be followed indefinitely Overexertion and emotional strain are to be avoided at all times and during periods of upper respiratory or other infections, the patient should be confined to bed until the fever and other symptoms have disappeared

Chapter VIII

THE RISK OF ANESTHESIA AND SURGICAL OPERATION IN PATIENTS WITH CORONARY HEART DISEASE

IT STILL is quite commonly believed that during anesthesia and surgical operations the heart is subjected to a considerably increased demand for work, but there is no evidence that this is actually the case The two greatest dangers to which a patient is exposed during an operation under anesthesia are anoxia and shock If these are avoided by proper anesthesia, minimizing of blood loss, and careful manipulation of tissues even prolonged and extensive surgical procedures do not increase the load on the heart as much as does moderate physical exertion Competent anesthesia is seldom attended by cyanosis or more than slight alterations in respiration, pulse rate, and blood pressure and changes of this kind are encountered no more frequently in patients who have organic heart disease with a satisfactory myocardial reserve than in normal individuals It therefore may be taken as a general rule that if anoxia and shock are avoided, patients with coronary artery disease who have been able to carry on normal daily activities without experiencing symptoms of myocardial or coronary insufficiency can tolerate general anesthesia and surgery with no more risk than a normal person There are a few exceptions to this rule, and these will be mentioned later

In estimating the risk of anesthesia and surgery in a patient who has coronary heart disease, careful inquiry concerning dyspnea or substernal pain on exertion, paroxysmal nocturnal dyspnea attacks of acute coronary failure, and former acute myocardial infarction is of much more impor

tance than are the cardiac findings on physical examination Certain findings, however, such as enlargement of the heart, gallop rhythm, and important disturbances of cardiac rhythm, may make it advisable to requestion the patient in order to be certain that the questions have been understood and truthfully answered The same consideration applies to the majority of electrocardiographic abnormalities, for, with few exceptions, an abnormal electrocardiogram in an individual who has had no symptoms of reduced myocardial or coronary reserve does not indicate an increased risk from anesthesia and surgery

In patients who have coronary artery disease with congestive heart failure or a history of paroxysmal cardiac dyspnea, a period of preoperative treatment is advisable, and the intensity and duration of this will be determined by the degree of failure and the urgency of the surgical condition A surgical emergency, such as acute appendicitis or a perforated peptic ulcer, will not permit delay, and the risk of immediate operation must be accepted If the patient has not been receiving digitalis in a situation of this kind ouabain or a suitable digitalis preparation should be administered by intravenous injection before beginning the anesthesia If ouabain is employed, the dose is usually 0 5 mg Additional injections of 0 1 to 0 25 mg may be given at intervals of four hours after the operation until a total of not more than 1 mg has been administered or if there is no need for great hurry in obtaining the maximum therapeutic effect the process of digitalization may be completed by intramuscular injections of one of the preparations designed for this purpose

When congestive heart failure is present and the surgical condition does not require immediate operation, rapid digitalization should be accomplished by the oral administration of digitalis or digitoxin or, if nausea and vomiting are present, by the intramuscular injection of a suitable prep-

aration If auricular fibrillation is present, the ventricular rate furnishes a helpful guide as to whether or not the maximum therapeutic effect of the drug has been obtained sufficient digitalis is given to reduce the rate, in the absence of fever and thyrotoxicosis, to approximately 70 beats per minute When normal rhythm is present, the heart rate is of no aid in estimating the degree of digitalization, and one must then prescribe the amount of the drug required by the average patient and be guided by the general clinical response The sodium chloride content of the diet should be strictly limited, and if edema is present one of the mercurial diuretics should be administered Surgery should be postponed, if at all possible, until all evidence of congestive failure has disappeared If this is done the patient can reasonably be expected to tolerate anesthesia and operation satisfactorily but if sufficient delay is impossible and adequate treatment cannot be carried out, surgery may be followed by a considerable increase in the degree of failure

Regardless of the adequacy of the preoperative treatment, patients who have had congestive failure before surgery must be watched closely during the period after operation Postoperative complications such as pulmonary embolism, atelectasis, pneumonia, and abdominal distention are not well borne and may be responsible for a return of or an increase in the manifestations of failure The mortality related to these complications is considerably greater in patients who have had congestive failure before operation than in normal individuals or in those who have coronary heart disease with little or no impairment of myocardial reserve

Digitalis also should be administered before operation to individuals with coronary artery disease who do not have congestive failure but who have experienced dyspnea on moderate exertion and who present such abnormal findings as enlargement of the heart, auricular fibrillation, auricular flutter, frequent premature beats, or electrocardiographic

changes consistent with the presence of coronary disease or ventricular strain Although these patients usually tolerate anesthesia and operation satisfactorily without preoperative digitalization the added strain of unforeseen postoperative complications may be sufficient to precipitate congestive heart failure The preparatory use of digitalis will improve the ability of the heart to withstand such an added strain so that postoperative mortality should not be significantly greater than in individuals who have a normal myocardial reserve The amount of the drug given should be sufficient to accomplish theoretical digitalization and the dosage schedule will be determined by the time available

Certain manifestations of coronary heart disease are liable to result in sudden death even under normal circumstances, and in patients who have one of these conditions the occurrence of only mild anoxia or shock during or after an operation may directly precipitate the changes which suddenly terminate life The conditions include angina pectoris former myocardial infarction or acute coronary failure and high grade or complete auriculoventricular block complicated by the Adams-Stokes syndrome In the first three of these conditions sudden death results from ventricular fibrillation or cardiac arrest, while death from the Adams Stokes syndrome is due to ventricular asystole In patients who have had angina pectoris myocardial infarction or acute coronary failure, the fall in blood pressure which accompanies surgical or postoperative shock may reduce coronary blood flow to such an extent that coronary failure or acute myocardial infarction results These conditions, if not suddenly fatal, may be responsible for the rapid development of congestive heart failure It therefore is essential that everything possible be done to avoid anoxia and a fall in blood pressure in patients who have one of these more dangerous clinical forms of coronary disease

The presence of serious coronary heart disease may in

fluence the surgeon considerably in deciding upon the type of operation to be done In patients who have recently had acute myocardial infarction or coronary failure or who are suffering from angina pectoris, paroxysmal cardiac dyspnea or Adams Stokes attacks, only unavoidable operations are performed, and the surgical procedure elected is the simplest one that will accomplish the desired result For example, the repair of abdominal or inguinal hernias is postponed as long as the condition can be controlled reasonably well by mechanical means Myomata of the uterus are treated by radiation and not by surgery and vagotomy is to be preferred to more extensive procedures for intractable peptic ulcer The aim of the surgeon should be to do as little as possible and still insure the relative comfort of the patient for the remainder of a limited life expectancy

The presence of coronary artery disease also influences the choice of the anesthetic agent Nitrous oxide oxygen and ethylene oxygen, supplemented by ether if necessary, are well tolerated when strict precautions are taken to avoid anoxia Chloroform and cyclopropane should not be used, and pentothal sodium should be employed with caution Whenever the patient has one of those conditions which are liable to cause sudden death, it is advisable to proceed as far as possible with local anesthesia The same conditions contraindicate the use of spinal anesthesia because this form of anesthesia often causes a decrease in blood pressure and a consequent reduction in coronary blood flow Acute coronary failure acute myocardial infarction or sudden death may result

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This Book

Coronary HEART DISEASE

By

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